NCBI PubMed www.pubmed.gov

A service of the National Library of Medicine and the National Institutes of Health

My NCBI [Sign In] [Regi

All Databases PubMed Nucleotide Protein Genome Structure OMIM PMC Journals Boo

Search PubMed for uj127 antibody Preview Go Cl

Limits Preview/Index History Clipboard Details

- Search History will be lost after eight hours of inactivity.
- To combine searches use # before search number, e.g., #2 AND #6.
- Search numbers may not be continuous; all searches are represented.
- Click on query # to add to strategy

About Entrez
NCBI Toolbar

Text Version

Entrez PubMed
Overview
Help | FAQ
Tutorials
New/Noteworthy
E-Utilities

PubMed Services
Journals Database
MeSH Database
Single Citation Matcher
Batch Citation Matcher
Clinical Queries
Special Queries
LinkOut
My NCBI

Related Resources
Order Documents
NLM Mobile
NLM Catalog
NLM Gateway
TOXNET
Consumer Health
Clinical Alerts
ClinicalTrials.gov
PubMed Central

Search	Most Recent Queries	Time	Result
#15	Search **uj127 antibody**	10:54:42	14
#14	Search **L1-11A antibody**	10:54:26	1
#12	Search **chCe7 antibody**	10:52:33	16
#10	Search **5G3 antibody**	10:51:38	4
#9	Search **#5 and cancer growth**	10:51:16	5
#7	Search **#5 and tumor growth**	10:49:38	9
#6	Search **antibody L1 antigen and proliferation**	10:15:56	6
#5	Search **antibody L1 antigen**	10:15:46	231
#3	Search **antibody UJ127**	09:56:01	14
#2	Search **L1CAM antibody UJ127**	09:55:36	1
#1	Search **L1CAM antibody**	09:55:03	104

Clear History

Welcome to DialogClassic Web(tm)

```
 Dialog level 05.11.05D
Last logoff:  17may06 15:30:02
Logon file001 19may06 09:59:24
          *** ANNOUNCEMENTS ***
                    ***
NEW FILES RELEASED
***Regulatory Affairs Journals (File 183)
***Index Chemicus (File 302)
***Inspec (File 202)

RESUMED UPDATING
***File 141, Reader's Guide Abstracts
                    ***
RELOADS COMPLETED
***File 516, D&B--Dun's Market Identifiers
***File 523, D&B European Dun's Market Identifiers
***File 531, American Business Directory
*** MEDLINE has been reloaded with the 2006 MeSH (Files 154 & 155)
*** The 2005 reload of the CLAIMS files (Files 340, 341, 942)
is now available online.
                                ***
DATABASES REMOVED
***File 196, FINDEX
***File 468, Public Opinion Online (POLL)
 Chemical Structure Searching now available in Prous Science Drug
Data Report (F452), Prous Science Drugs of the Future (F453),
IMS R&D Focus (F445/955), Pharmaprojects (F128/928), Beilstein
Facts (F390), Derwent Chemistry Resource (F355) and Index Chemicus
(File 302).
                    ***
 >>>For the latest news about Dialog products, services, content<<<
 >>>and events, please visit What's New from Dialog at <<<
 >>>http://www.dialog.com/whatsnew/.  You can find news about<<<
 >>>a specific database by entering HELP NEWS <file number>.<<<
 * * *

File   1:ERIC 1966-2006/Apr (c) format only 2006 Dialog

      Set  Items  Description
      ---  -----  -----------
Cost is in DialUnits
?

B 155, 159, 10, 203, 35, 5, 467, 73, 434, 34
       19may06 10:00:25 User290558 Session D42.1
            $0.81    0.232 DialUnits File1
     $0.81  Estimated cost File1
     $0.53  INTERNET
     $1.34  Estimated cost this search
     $1.34  Estimated total session cost   0.232 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R) 1951-2006/May 23
         (c) format only 2006 Dialog
  File 159:Cancerlit 1975-2002/Oct
         (c) format only 2002 Dialog
 *File 159: Cancerlit is no longer updating.
Please see HELP NEWS159.
  File  10:AGRICOLA 70-2006/Apr
```

```
        (c) format only 2006 Dialog
  File 203:AGRIS 1974-2006/Mar
        Dist by NAL, Intl Copr. All rights reserved
  File  35:Dissertation Abs Online 1861-2006/Apr
        (c) 2006 ProQuest Info&Learning
  File   5:Biosis Previews(R) 1969-2006/May W2
        (c) 2006 BIOSIS
  File 467:ExtraMED(tm) 2000/Dec
        (c) 2001 Informania Ltd.
 *File 467: F467 will close on February 1, 2006.                    7.
  File  73:EMBASE 1974-2006/May 19
        (c) 2006 Elsevier Science B.V.
  File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
        (c) 1998 Inst for Sci Info
  File  34:SciSearch(R) Cited Ref Sci 1990-2006/May W2
        (c) 2006 Inst for Sci Info

      Set  Items  Description
      ---  -----  -----------
?

S ((L1CAM) OR (L1 (N) CELL (N) ADHESION) AND ANTIBODY)
             475  L1CAM
           65124  L1
        11105775  CELL
          510366  ADHESION
             447  L1(N)CELL(N)ADHESION
         1751004  ANTIBODY
      S1     526  ((L1CAM) OR (L1 (N) CELL (N) ADHESION) AND ANTIBODY)
?

S ((TUMOR OR TUMOUR OR CANCER OR NEOPLASIA OR CARCINOMA) AND (GROWTH OR PROLIFERATIO
Processing
Processed  10 of  10 files ...
Completed processing all files
         3228374  TUMOR
          384028  TUMOUR
         3443099  CANCER
          136893  NEOPLASIA
         1732409  CARCINOMA
         4409277  GROWTH
          896535  PROLIFERATION
      S2  971036  ((TUMOR OR TUMOUR OR CANCER OR NEOPLASIA OR CARCINOMA)
                  AND (GROWTH OR PROLIFERATION))
?


Set     Items   Description
S1        526   ((L1CAM) OR (L1 (N) CELL (N) ADHESION) AND ANTIBODY)
S2     971036   ((TUMOR OR TUMOUR OR CANCER OR NEOPLASIA OR CARCINOMA) AND
            (GROWTH OR PROLIFERATION))
?

S S1 AND S2
             526  S1
          971036  S2
      S3      29  S1 AND S2
?

RD S3
```

S4 13 RD S3 (unique items)
?

TYPE S4/MEDIUM,K/1-13

4/K/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

19910302 PMID: 16424028
Efficient inhibition of intra-peritoneal tumor growth and dissemination of human ovarian carcinoma cells in nude mice by anti-L1-cell adhesion molecule monoclonal antibody treatment.
Arlt Matthias J E; Novak-Hofer Ilse; Gast Daniela; Gschwend Verena; Moldenhauer Gerhard; Grunberg Jurgen; Honer Michael; Schubiger P August; Altevogt Peter; Kruger Achim
Klinikum rechts der Isar der Technischen Universitat Munchen, Institut fur Experimentelle Onkologie und Therapieforschung, Ismaninger Strasse 22, D-81675 Munich, Germany.
Cancer research (United States) Jan 15 2006, 66 (2) p936-43, ISSN 0008-5472--Print Journal Code: 2984705R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Efficient inhibition of intra-peritoneal tumor growth and dissemination of human ovarian carcinoma cells in nude mice by anti- L1 - cell adhesion molecule monoclonal antibody treatment.
The L1 cell adhesion molecule is implicated in the control of proliferation , migration, and invasion of several tumor cell types in vitro. Recently, L1 overexpression was found to correlate with tumor progression of ovarian carcinoma , one of the most common causes of cancer -related deaths in gynecologic malignant diseases. To evaluate L1 as a potential target for ovarian cancer therapy, we investigated the effects of anti-L1 monoclonal antibodies (chCE7 and L1-11A) on proliferation and migration of L1-positive human SKOV3ip ovarian carcinoma cells in vitro and the therapeutic efficacy of L1-11A against i.p. SKOV3ip tumor growth in nude mice. In vitro, both anti-L1 antibodies efficiently inhibited the proliferation of SKOV3ip cells as well as other L1-expressing tumor cell lines (renal carcinoma , neuroblastoma, and colon carcinoma). On two cell lines, hyper-cross-linking of L1-11A with a secondary antibody was necessary for significant inhibition of proliferation , indicating that cross-linking of L1 is required for the antiproliferative effect. L1-negative prostate carcinoma cells were not influenced by antibody treatment. Biweekly treatment of ovarian carcinoma -bearing mice with L1-11A led to a dose-dependent and significant reduction of tumor burden (up to -63.5%) and ascites formation (up to -75%). This effect was associated with reduced proliferation within the tumors. L1-directed antibody -based inhibition of peritoneal growth and dissemination of human ovarian carcinoma cells represents important proof-of-principle for the development of a new therapy against one...
Descriptors: *Antibodies, Monoclonal--therapeutic use--TU; * Carcinoma --pathology--PA; *Neural Cell Adhesion Molecule L1--immunology--IM; *Ovarian Neoplasms--pathology--PA; *Peritoneal Neoplasms...
; Animals; Carcinoma --genetics--GE; Carcinoma --therapy--TH; Cell Movement; Cell Proliferation ; Disease Progression; Humans; Mice; Mice, Nude; Neural Cell Adhesion Molecule L1--physiology--PH; Ovarian Neoplasms

...

4/K/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15329279 PMID: 15709188
The L1 cell adhesion molecule is induced in renal cancer cells and correlates with metastasis in clear cell carcinomas.
Allory Yves; Matsuoka Yasuko; Bazille Celine; Christensen Erik Ilso; Ronco Pierre; Debiec Hanna
INSERM U489, Tenon Hospital (Assistance Publique-Hopitaux de Paris) and Paris 6 University, 4 rue de la Chine, Paris 70520, France.
Clinical cancer research - an official journal of the American Association for Cancer Research (United States) Feb 1 2005, 11 (3) p1190-7, ISSN 1078-0432--Print Journal Code: 9502500
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

The L1 cell adhesion molecule is induced in renal cancer cells and correlates with metastasis in clear cell carcinomas.
PURPOSE: The L1 cell adhesion molecule is overexpressed in many human carcinomas. The objectives of the study were to provide...
... kidney and renal tumors of diverse histopathologic origin, then we studied L1 expression together with tumor stage, grade, molecular prognostic biomarkers, and metastatic behavior. RESULTS: In normal kidney, L1 immunoreactive with...

...of 88 papillary RCC (papRCC)]. Both in ccRCC and papRCC, L1 reacted only with the antibody to the extracellular domain, suggesting that the protein was truncated. In these carcinomas, L1 expression was strongly correlated with Ki-67 proliferation index (ccRCC, P = 0.0059; papRCC, P = 0.0039), but only in ccRCC, the presence...

... metastasis (P = 0.0121). This risk was higher if cyclin D1 was concurrently absent in tumor cells (P < 0.0001). The L1(+)/cyclin D1(-) profile was an independent prognostic factor of...
Descriptors: *Adenocarcinoma, Clear Cell--pathology--PA; * Carcinoma , Renal Cell--pathology--PA; *Kidney Neoplasms--pathology--PA; *Neural Cell Adhesion Molecule L1--genetics--GE...; Cell--genetics--GE; Adenocarcinoma, Clear Cell--metabolism--ME; Adolescent; Adult; Aged; Aged, 80 and over; Carcinoma , Renal Cell--genetics--GE; Carcinoma , Renal Cell--metabolism --ME; Comparative Study; Cyclin D1--analysis--AN; Gene Expression Regulation, Neoplastic; Humans...

4/K/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15260591 PMID: 15447976
L1CAM, INP10, P-cadherin, tPA and ITGB4 over-expression in malignant pleural mesotheliomas revealed by combined use of cDNA and tissue microarray.
Kettunen E; Nicholson A G; Nagy B; Wikman H; Seppanen J K; Stjernvall T; Ollikainen T; Kinnula V; Nordling S; Hollmen J; Anttila S; Knuutila S

Department of Pathology, Haartman Institute and HUSLAB, University of Helsinki, Helsinki University Central Hospital, Helsinki, Finland.
Carcinogenesis (England) Jan 2005, 26 (1) p17-25, ISSN 0143-3334--Print Journal Code: 8008055
Publishing Model Print-Electronic
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

L1CAM , INP10, P-cadherin, tPA and ITGB4 over-expression in malignant pleural mesotheliomas revealed by combined...

Malignant pleural mesothelioma (MM) is a rare tumour with high mortality, which can exhibit various morphologies classified as epithelioid, biphasic and sarcomatoid subtypes...

...in these tumours, we studied gene expression patterns by combined use of cDNA arrays and tumour tissue microarrays (TMA). Deregulation of the expression of 588 cancer -related genes was screened in 16 MM comprising all three subtypes and compared with references...

...and tissue-type plasminogen activator (tPA) in sarcomatoid MM and neural cell adhesion molecule L1 (L1CAM) and INP10 in biphasic MM. Immunohistochemistry on TMA containing 47 MM (26 epithelioid, 15 sarcomatoid and six biphasic) was performed for five proteins, ITGB4, P-cadherin, tPA, INP10 and L1CAM . INP10 expression was increased in MM in general compared with normal mesothelium, while increased expression of P-cadherin, L1CAM and ITGB4 was more specific in MMs exhibiting an epithelioid growth pattern. The over-expression of tPA was more frequent in epithelioid MM despite higher mRNA...

...and biphasic MM. We conclude that several proteins, associated with cell adhesion either directly (ITGB4, L1CAM , P-cadherin) or as a regulatory factor (INP10), are differentially expressed in MM. In particular...

4/K/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15133655 PMID: 15361835
Identification of cell surface and secreted proteins essential for tumor cell survival using a genetic suppressor element screen.

Gelman Marina S; Ye X Katherine; Stull Robert; Suhy David; Jin Liang; Ng Dean; Than Bruce; Ji May; Pan Alison; Perez Paul; Sun Yan; Yeung Patricia; Garcia Luz Maria; Harte Rachel; Lu Yan; Lamar Elizabeth; Tavassoli Roya; Kennedy Scot; Osborn Stephen; Chin Daniel J; Meshaw Kay; Holzmayer Tatyana A; Axenovich Sergey A; Abo Arie
PPD Discovery, Inc., 1505 O'Brien Drive, Menlo Park, CA 94025, USA.
Oncogene (England) Oct 21 2004, 23 (49) p8158-70, ISSN 0950-9232--Print Journal Code: 8711562
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Identification of cell surface and secreted proteins essential for tumor cell survival using a genetic suppressor element screen.

Survival factors play critical roles in regulating cell growth in

normal and cancer cells. We designed a genetic screen to identify survival factors which protect tumor cells from apoptosis. A retroviral expression library of random cDNA fragments was constructed from cancer cells and used to transduce the colon carcinoma cell line HCT116. Recipient cells were functionally selected for induction of caspase 3-mediated apoptosis...

... derived from the same genes. Our data suggest requirement for the cell surface targets IGF2R, L1CAM and SLC31A1 in tumor cell growth in vitro, and suggests that IGF2R is required for xenograft tumor growth in a mouse model.
; Animals; Caspases--physiology--PH; Cell Division; Cell Line, Tumor ; Cell Survival; Humans; Mice; Neoplasm Transplantation; RNA, Small Interfering--pharmacology--PD; Receptor, IGF Type 2...

4/K/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15091811 PMID: 15446585
Gene expression profiling reveals unique molecular subtypes of Neurofibromatosis Type I-associated and sporadic malignant peripheral nerve sheath tumors.
Watson Mark A; Perry Arie; Tihan Tarik; Prayson Richard A; Guha Abhijit; Bridge Julia; Ferner Rosalie; Gutmann David H
Department of Pathology and Immunology, Washington University School of Medicine, St Louis, MO 63110, USA. watsonm@pathbox.wustl.edu
Brain pathology (Zurich, Switzerland) (Switzerland) Jul 2004, 14 (3) p297-303, ISSN 1015-6305--Print Journal Code: 9216781
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

... analyzed. This signature corresponded to relative overexpression of transcripts associated with neuroglial differentiation (NCAM, MBP, L1CAM , P1P) and relative down-regulation of proliferation and growth factor associated transcripts (IGF2, FGFR1, MDK, Ki67). All tumors with this gene expression signature lacked expression of EGFR and all but one tumor were derived from patients with NF1. However, there were no other obvious associations with histological grade, tumor site, metastasis, recurrence, age, or patient survival. We conclude that distinct molecular classes of MPNST...
...; Humans; Middle Aged; Nerve Sheath Neoplasms--complications--CO; Oligonucleotide Array Sequence Analysis; Prognosis; Receptor, Epidermal Growth Factor--biosynthesis--BI; Receptor, Epidermal Growth Factor --genetics--GE; Research Support, Non-U.S. Gov't
Enzyme No.: EC 2.7.1.112 (Receptor, Epidermal Growth Factor)
Chemical Name: Receptor, Epidermal Growth Factor

4/K/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14424637 PMID: 12892712
Identification of potential anticancer drug targets through the selection of growth-inhibitory genetic suppressor elements.

Primiano Thomas; Baig Mirza; Maliyekkel Anil; Chang Bey Dih; Fellars Stacey; Sadhu Justin; Axenovich Sergey A; Holzmayer Tatyana A; Roninson Igor B
Department of Molecular Genetics, University of Illinois at Chicago, Chicago, IL 60607, USA.
Cancer cell (United States) Jul 2003, 4 (1) p41-53, ISSN 1535-6108 --Print Journal Code: 101130617
Contract/Grant No.: R01 CA62099; CA; NCI; R01 CA95727; CA; NCI; R21 CA76908; CA; NCI
Publishing Model Print; Erratum in Cancer Cell. 2003 Nov;4(5) 415
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Identification of potential anticancer drug targets through the selection of growth -inhibitory genetic suppressor elements.

To identify human genes required for tumor cell growth , transcriptome-scale selection was used to isolate genetic suppressor elements (GSEs) inhibiting breast carcinoma cell growth . Growth -inhibitory GSEs (cDNA fragments that counteract their cognate gene) were selected from 57 genes, including known positive regulators of cell growth or carcinogenesis as well as genes that have not been previously implicated in cell proliferation . Many GSE-cognate genes encode transcription factors (such as STAT and AP-1) and signal...

... Monoclonal antibodies against a cell surface protein identified by GSE selection, neural cell adhesion molecule L1CAM , strongly inhibited the growth of several tumor cell lines but not of untransformed cells. Hence, selection for growth -inhibitory GSEs allows one to find potential targets for new anticancer drugs.

...; Gov't; Research Support, U.S. Gov't, P.H.S.; Transcription Factors --immunology--IM; Tumor Cells, Cultured

4/K/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11556362 PMID: 9371782

The phosphorylation state of the FIGQY tyrosine of neurofascin determines ankyrin-binding activity and patterns of cell segregation.

Tuvia S; Garver T D; Bennett V
Howard Hughes Medical Institute and Department of Cell Biology, Duke University Medical Center, Durham, NC 27710, USA.
Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Nov 25 1997, 94 (24) p12957-62, ISSN 0027-8424--Print Journal Code: 7505876
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

... regulation of cell interactions through modulation of ankyrin binding to neurofascin, a member of the L1CAM family of nervous system cell adhesion molecules. The phosphorylation state of the conserved FIGQY tyrosine...

... for the patterning of cell contact based on external signals that

regulate tyrosine phosphorylation of L1CAM members and modulate their binding to ankyrin.
Descriptors: *Ankyrins--metabolism--ME; *Cell Adhesion Molecules --metabolism--ME; *Nerve Growth Factors--metabolism--ME; *Tyrosine --metabolism--ME...; Adhesion Molecules--chemistry--CH; Cell Aggregation; Cell Separation; Cytoplasm--metabolism--ME; Molecular Sequence Data; Nerve Growth Factors--chemistry--CH; Phosphorylation; Protein Binding; Tumor Cells, Cultured
Chemical Name: Ankyrins; Cell Adhesion Molecules; Nerve Growth Factors; Tyrosine

4/K/8 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014544177 BIOSIS NO.: 200300499205
L1CAM is a novel potential target for cancer therapy.
AUTHOR: Primiano Thomas (Reprint); Baig Mirza (Reprint); Roninson Igor B (Reprint)
AUTHOR ADDRESS: Department of Molecular Genetics, University of Illinois-Chicago, Chicago, IL, 60607, USA**USA
JOURNAL: Proceedings of the American Association for Cancer Research Annual Meeting 44 p1351-1352 July 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 94th Annual Meeting of the American Association for Cancer Research Washington, DC, USA July 11-14, 2003; 20030711
ISSN: 0197-016X
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

L1CAM is a novel potential target for cancer therapy.
DESCRIPTORS:
...MAJOR CONCEPTS: Tumor Biology
...ORGANISMS: human colon carcinoma cells...
...human cervical carcinoma cells...

...human breast carcinoma cells...

...human breast carcinoma cells...
DISEASES: breast carcinoma --...

...cervical carcinoma --...

...colon carcinoma --
...MESH TERMS: Carcinoma (MeSH...

... Carcinoma (MeSH...

... Carcinoma (MeSH)
CHEMICALS & BIOCHEMICALS: ... growth -inhibitory genetic suppressor element {GSE...

...immunoglobulin-like domain of cell adhesion molecule L1 { L1CAM }--
METHODS & EQUIPMENT: cancer therapy...
MISCELLANEOUS TERMS: tumor cell growth ;

4/K/9 (Item 1 from file: 73)

DIALOG(R)File 73:EMBASE

13013899 EMBASE No: 2005077852
Molecular profiling of malignant peripheral nerve sheath tumors associated with neurofibromatosis type 1, based on large-scale real-time RT-PCR
Levy P.; Vidaud D.; Leroy K.; Laurendeau I.; Wechsler J.; Bolasco G.; Parfait B.; Wolkenstein P.; Vidaud M.; Bieche I.
I. Bieche, Laboratoire de Genetique Moleculaire, Faculte Sci. Pharmaceut./Biologiques, Universite Paris V, Paris France
AUTHOR EMAIL: ivan.bieche@univ-paris5.fr
Molecular Cancer (MOL. CANCER) (United Kingdom) 15 JUL 2004, 3/- (13p)
CODEN: MCOAC ISSN: 1476-4598
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 59

...upregulated and 12 were downregulated in MPNSTs. The altered genes were mainly involved in cell proliferation (MKI67, TOP2A, CCNE2), senescence (TERT, TERC), apoptosis (BIRC5/Survivin, TP73) and extracellular matrix remodeling (MMP13...
...Gli signaling pathway (DHH, PTCH2). Several of the down-regulated genes were Schwann cell-specific (L1CAM , MPZ, S100B, SOX10, ERBB3) or mast cell-specific (CMA1, TPSB), pointing to a depletion and...
DRUG DESCRIPTORS:
...endogenous compound--ec; protein S100B--endogenous compound--ec; transcription factor Sox10--endogenous compound--ec; epidermal growth factor receptor 3--endogenous compound--ec; sonic hedgehog protein --endogenous compound--ec; gene product--endogenous...
MEDICAL DESCRIPTORS:
*nerve sheath tumor --etiology--et; *neurofibromatosis
...nerve; real time polymerase chain reaction; carcinogenesis; gene expression; gene control; upregulation; down regulation; cell proliferation ; senescence; apoptosis; extracellular matrix; signal transduction; Schwann cell; cell specificity; mast cell; cell differentiation; malignant...
...CAS REGISTRY NO.: 4 (tissue inhibitor of metalloproteinase 4); 357701-89-4 (protein S100B); 497121-54-7 (epidermal growth factor receptor 3)

4/K/10 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

11144141 EMBASE No: 2001159737
Expression of neural cell adhesion molecules (polysialylated form of neural cell adhesion molecule and L1-cell adhesion molecule) on resected small cell lung cancer specimens: In relation to proliferation state
Miyahara R.; Tanaka F.; Nakagawa T.; Matsuoka K.; Isii K.; Wada H.
Dr. H. Wada, Kyoto University, Department of Respiratory Surgery, 53 Kawahara-chou Shogoin Sakyou-ku, Kyoto 606-01 Japan
AUTHOR EMAIL: wadah@kuhp.kyoto-u.ac.jp
Journal of Surgical Oncology (J. SURG. ONCOL.) (United States) 2001, 77/1 (49-54)
CODEN: JSONA ISSN: 0022-4790
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 25

Expression of neural cell adhesion molecules (polysialylated form of neural cell adhesion molecule and L1 - cell adhesion molecule) on resected small cell lung cancer specimens: In relation to proliferation state

...Alteration of homotypic cell-cell adhesion has been suggested to play an important role in tumor progression. The present study examined the relationship between neural cell adhesion molecules and state of proliferation of small cell lung cancer (SCLC) cells. Methods: Seventeen surgically resected specimens of SCLC were immunohistochemically examined, by using monoclonal...

...and NCAM a marker for SCLC. L1-CAM may be synthesized independent of state of proliferation of individual tumor cell and may affect clinical feature of SCLC. (c) 2001 Wiley-Liss, Inc.
DRUG DESCRIPTORS:
monoclonal antibody ; tumor marker--endogenous compound--ec; polysialic acid--endogenous compound--ec; unclassified drug
MEDICAL DESCRIPTORS:
*lung small cell cancer
protein expression; cell adhesion; tumor growth ; cancer cell; cell proliferation ; immunohistochemistry; antibody labeling; prognosis; protein synthesis; clinical feature; human; male; female; clinical article; human tissue; adolescent; aged...
SECTION HEADINGS:
005 General Pathology and Pathological Anatomy
016 Cancer
029 Clinical and Experimental Biochemistry

4/K/11 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

13624839 Genuine Article#: 898SZ No. References: 59
Title: Identification of L1CAM, Jagged2 and neuromedin U as ovarian cancer-associated antigens
Author(s): Euer NI; Kaul S; Deissler H; Mobus VJ; Zeillinger R; Weidle UH (REPRINT)
Corporate Source: Roche Diagnost GmbH,Pharma Res,Nonnenwald 2/D-82372 Penzberg//Germany/ (REPRINT); Roche Diagnost GmbH,Pharma Res,D-82372 Penzberg//Germany/; Univ Heidelberg,Womens Hosp,D-6900 Heidelberg//Germany/; Univ Ulm,Sch Med, Dept Obstet & Gynecol,Ulm//Germany/; Stadt Kliniken,Dept Obstet & Gynecol,Frankfurt//Germany/; Med Univ Vienna,Dept Obstet & Gynecol, Div Gynecol,Vienna//Austria/(ulrich.weidle@roche.com)
Journal: ONCOLOGY REPORTS, 2005, V13, N3 (MAR), P375-387
ISSN: 1021-335X Publication date: 20050300
Publisher: PROFESSOR D A SPANDIDOS, 1, S MERKOURI ST, EDITORIAL OFFICE,, ATHENS 116 35, GREECE
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

Title: Identification of L1CAM , Jagged2 and neuromedin U as ovarian cancer -associated antigens
Abstract: In order to identify tumor -associated genes of ovarian carcinoma , we have investigated the transcriptional profile of 11 ovarian tumor cell lines and 2 immortalized ovarian surface epithelial cell lines (IOSE) derived from normal ovarian...

...up-regulated and 165 were down-regulated in at least 7 out of 11 ovarian tumor cell lines in comparison to the transcriptional profile of the IOSE cell lines with a...

...receptors and secreted proteins as possible markers for diagnosis and targets for therapy of ovarian carcinoma . We have identified the transmembrane Notch ligand Jagged2, cell adhesion molecule LICAM and the secreted...

...borderline tumors to a lesser extent, and very rarely in ovarian non-epithelial types of cancer . Further analysis of LICAM revealed that a splice variant lacking exons 2 and 27 is predominantly expressed in ovarian carcinoma cell lines DW and GG. Functional investigation of stable Delta(2,27)LICAM transfectants of the ovarian tumor cell line OV-MZ-2 revealed significantly stronger adhesion to laminin in comparison to mock...
...Identifiers--FACTOR-BINDING PROTEIN-2; ADHESION MOLECULE L1; TUMOR -SUPPRESSOR GENE; CARCINOMA CELL-LINES; HEPATOCELLULAR- CARCINOMA ; EXTRACELLULAR-MATRIX; NERVOUS-SYSTEM; EARLY STEP; EXPRESSION; GROWTH

4/K/12 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

02733226 Genuine Article#: LZ700 No. References: 49
Title: EXPRESSION OF L1 CELL-ADHESION MOLECULE IS ASSOCIATED WITH LYMPHOMA GROWTH AND METASTASIS
Author(s): KOWITZ A; KADMON G; VERSCHUEREN H; REMELS L; DEBAETSELIER P; HUBBE M; SCHACHNER M; SCHIRRMACHER V; ALTEVOGT P
Corporate Source: GERMAN CANC RES CTR,INST IMMUNOL & GENET,NEUENHEIMER FELD 280/W-6900 HEIDELBERG//GERMANY/; GERMAN CANC RES CTR,INST IMMUNOL & GENET,NEUENHEIMER FELD 280/W-6900 HEIDELBERG//GERMANY/; PASTEUR INST VAN BRABANT/BRUSSELS//BELGIUM/; SWISS FED INST TECHNOL,DEPT NEUROBIOL/CH-8092 ZURICH//SWITZERLAND/
Journal: CLINICAL & EXPERIMENTAL METASTASIS, 1993, V11, N5 (SEP), P419-429
ISSN: 0262-0898
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

Title: EXPRESSION OF L1 CELL - ADHESION MOLECULE IS ASSOCIATED WITH LYMPHOMA GROWTH AND METASTASIS
...Abstract: recently also been identified on leucocytes. We have investigated the expression of L1 on hematopoietic tumor cell lines and found that several tumors including the ESb-MP lymphoma are positive for...
...L1low expression variants. Syngeneic DBA/2 mice injected subcutaneously with L1low clones showed faster primary tumor growth , developed visceral metastases significantly faster and died earlier than animals carrying L1high wt cells. L1high...

...metastatic capacity and a malignancy similar to the wt cells. Expression of L1 on the tumor variants and revertants correlated directly with their homotypic aggregation behaviour in vitro. L1 expression correlated...

...malignant potential of the lymphoma cells, presumably by interfering with cell-cell interactions critical for tumor growth and dissemination.
...Research Fronts: RECEPTOR; ALLERGIC CUTANEOUS INFLAMMATION INVIVO)

```
  91-2216 001   (NEURAL CELL-ADHESION MOLECULES; DIFFERENTIAL EXPRESSION;
    NEURONAL  GROWTH  CONE)
  91-4842 001   (T-CELL RECEPTOR EXPRESSION; MURINE THYMUS; ANTI-CD4
    ANTIBODY )
  91-5178 001   (MEMBRANE GLYCOPROTEIN; POSTTRANSLATIONAL REGULATION OF IGM
    EXPRESSION; CELL-SURFACE ANTIGEN; COMPLEX OLIGOSACCHARIDES)


  4/K/13     (Item 3 from file: 34)
DIALOG(R)File  34:SciSearch(R) Cited Ref Sci
(c) 2006 Inst for Sci Info. All rts. reserv.

01164887   Genuine Article#: GB048   No. References: 23
 Title: THE 200 220 KDA ANTIGEN RECOGNIZED BY MONOCLONAL-ANTIBODY (MAB)
   UJ127.11 ON NEURAL TISSUES AND TUMORS IS THE HUMAN-L1 ADHESION MOLECULE
Author(s): PATEL K; KIELY F; PHIMISTER E; MELINO G; RATHJEN F; KEMSHEAD JT
Corporate Source: FRENCHAY HOSP,IMPERIAL CANC RES FUND,PAEDIAT & NEUROONCOL
    GRP/BRISTOL BS16 1LE/AVON/ENGLAND/; FRENCHAY HOSP,IMPERIAL CANC RES
    FUND,PAEDIAT & NEUROONCOL GRP/BRISTOL BS16 1LE/AVON/ENGLAND/; CTR MOLEC
    NEUROBIOL/D-2000 HAMBURG 20//FED REP GER/; UNIV TOR
    VERGATA,DIPARTIMENTO MED SPERIMENTALE 2/I-00173 ROME//ITALY/
Journal: HYBRIDOMA, 1991, V10, N4, P481-491
Language: ENGLISH   Document Type: ARTICLE   (Abstract Available)

 Title: THE 200 220 KDA ANTIGEN RECOGNIZED BY MONOCLONAL- ANTIBODY  (MAB)
   UJ127.11 ON NEURAL TISSUES AND TUMORS IS THE HUMAN-L1 ADHESION MOLECULE
...Abstract: raised against 16 week human fetal brain, recognizes an
    antigen present primarily on normal and  tumor  tissues derived from
    the neuroectoderm.  The antigen has previously been identified as a
    220/240...

...by immunoprecipitation studies.  We show here, that the 220/240 kDa
    antigen is the human  L1  cell  adhesion  molecule and by Western
    blot analysis actually has a calculated molecular weight of between
    200-220 kDa.  Immunocytochemical studies with UJ127.11 and an  antibody
     (5G3) recently utilized to isolate human L1 from brain indicate that
    both reagents have very...
Research Fronts: 89-0577 001   (PLATELET-DERIVED  GROWTH -FACTOR; PDGF
    RECEPTOR; ONCOGENE EXPRESSION; C-ERBB-2 AMPLIFICATION IN HUMAN-BREAST
    CARCINOMA ; NON-SMALL CELL LUNG- CANCER)
?


Set     Items   Description
S1        526   ((L1CAM) OR (L1 (N) CELL (N) ADHESION) AND ANTIBODY)
S2     971036   ((TUMOR OR TUMOUR OR CANCER OR NEOPLASIA OR CARCINOMA) AND
             (GROWTH OR PROLIFERATION))
S3         29   S1 AND S2
S4         13   RD S3  (unique items)
?

S (UJ127 AND ANTIBODY)
              76  UJ127
         1751004  ANTIBODY
      S5      50  (UJ127 AND ANTIBODY)
?

RD S5
      S6      19  RD S5  (unique items)
?
```

```
S (S6 AND (PROLIFERATION OR GROWTH))
              19  S6
          896535  PROLIFERATION
         4409277  GROWTH
      S7       3  (S6 AND (PROLIFERATION OR GROWTH))
?

RD S7
      S8       3  RD S7  (unique items)
?

TYPE S8/MEDIUM,K/1-3
```

8/K/1 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

08667246 PMID: 2177080

Establishment and characterization of a primitive neuroectodermal tumor of bone continuous cell line (LAP-35).

Bagnara G P; Serra M; Giovannini M; Badiali M; Stella M; Montaldi A; Granchi D; Paolucci P; Rocchi P; Pession A; et al
G. Prodi Interdepartmental Center for Cancer Research, Bologna, Italy.

International journal of cell cloning (UNITED STATES) Nov 1990, 8 (6) p409-24, ISSN 0737-1454--Print Journal Code: 8308172
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

... year-old female. The neural character of the cell line was documented by the spontaneous growth of neurites and by the presence of several neural markers, including neuron-specific enolase (NSE), S-100 protein, neurofilaments, chromogranin A, synaptophysin and positivity to monoclonal antibodies UJ127 .11, UJ13A, UJ181.4. Cell-sorter analysis showed a high expression of nerve growth factor receptor (NGFr) and major histocompatibility complex class I-related molecules. A unique cytogenetic profile...

; Animals; Cell Division--physiology--PH; Child; Flow Cytometry; Fluorescent Antibody Technique; Genes, myc--genetics--GE; Humans; Immunohistochemistry; Karyotyping; Mice; Mice, Nude; Microscopy, Electron; Neoplasm Transplantation

8/K/2 (Item 1 from file: 159)

DIALOG(R)File 159:Cancerlit

01715070 PMID: 89650834

MONOCLONAL ANTIBODIES AND DIAGNOSIS OF BRAIN NEOPLASMS.

McLendon; Vick; Bigner; Bigner
Duke Univ. Medical Center, Durham, NC

Immunol Ser 1988, 39 p31-66,
Document Type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
Languages: ENGLISH
Main Citation Owner: NOTNLM
Record type: Completed

Neural antigens identified by biochemical means and selected monoclonal antibody (MAb)-defined neural antigens are reviewed. The first category includes S-100 protein, intermediate filament...

... enolase, glutamine synthetase, alpha-2 glycoprotein, and gangliosides. The second category includes the MAbs UJ13A, UJ127 .11, UJ181.4, and antiglioma MAbs. As diagnostic pathology incorporates the techniques of cell culture...

... of molecular genetics, MAbs will be used to detect oncogene products, such as the epidermal growth factor produced by the c-erb b oncogene, which is increased in gliomas. The development...

8/K/3 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

06790656 EMBASE No: 1997072158
Development of Hassall's bodies of the thymus in humans and other vertebrates (especially mammals) under physiological and pathological conditions: Immunocytochemical, electronmicroscopic and in vitro observations
Bodey B.; Kaiser H.E.
B. Bodey, 15745 Saticoy Street, Van Nuys, CA 91406 United States
In Vivo (IN VIVO) (Greece) 1997, 11/1 (61-85)
CODEN: IVIVE ISSN: 0258-851X
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 335

...layer of the HBs reacted positively with medium to strong intensity when stained with MoABs UJ127 .11, J1153, A2B5, 215.D11, and 275.G7. These results further suggest that HBs are...

...various non-lymphatic thymic cells participating in the determination of the particular physiological activities, progressive growth , and the terminal cell differentiation within the HBs.
DRUG DESCRIPTORS:
acid glycosaminoglycan--endogenous compound--ec; keratin--endogenous compound--ec; membrane antigen--endogenous compound--ec; monoclonal antibody ; nonhistone protein--endogenous compound--ec; polyclonal antibody
?

```
Set     Items   Description
S1        526   ((L1CAM) OR (L1 (N) CELL (N) ADHESION) AND ANTIBODY)
S2     971036   ((TUMOR OR TUMOUR OR CANCER OR NEOPLASIA OR CARCINOMA) AND
              (GROWTH OR PROLIFERATION))
S3         29   S1 AND S2
S4         13   RD S3  (unique items)
S5         50   (UJ127 AND ANTIBODY)
S6         19   RD S5  (unique items)
S7          3   (S6 AND (PROLIFERATION OR GROWTH))
S8          3   RD S7  (unique items)
?

S (5G3 AND ANTIBODY)
              23  5G3
```

```
         1751004  ANTIBODY
      S9      17  (5G3 AND ANTIBODY)
?


Set     Items   Description
S1        526   ((L1CAM) OR (L1 (N) CELL (N) ADHESION) AND ANTIBODY)
S2     971036   ((TUMOR OR TUMOUR OR CANCER OR NEOPLASIA OR CARCINOMA) AND
             (GROWTH OR PROLIFERATION))
S3         29   S1 AND S2
S4         13   RD S3  (unique items)
S5         50   (UJ127 AND ANTIBODY)
S6         19   RD S5  (unique items)
S7          3   (S6 AND (PROLIFERATION OR GROWTH))
S8          3   RD S7  (unique items)
S9         17   (5G3 AND ANTIBODY)
?

S (S9 AND (PROLIFERATION AND GROWTH))
             17  S9
         896535  PROLIFERATION
        4409277  GROWTH
     S10       0  (S9 AND (PROLIFERATION AND GROWTH))
?


Set     Items   Description
S1        526   ((L1CAM) OR (L1 (N) CELL (N) ADHESION) AND ANTIBODY)
S2     971036   ((TUMOR OR TUMOUR OR CANCER OR NEOPLASIA OR CARCINOMA) AND
             (GROWTH OR PROLIFERATION))
S3         29   S1 AND S2
S4         13   RD S3  (unique items)
S5         50   (UJ127 AND ANTIBODY)
S6         19   RD S5  (unique items)
S7          3   (S6 AND (PROLIFERATION OR GROWTH))
S8          3   RD S7  (unique items)
S9         17   (5G3 AND ANTIBODY)
S10         0   (S9 AND (PROLIFERATION AND GROWTH))
?

RD S9
     S11       5  RD S9  (unique items)
?

TYPE S11/MEDIUM,K/1-5

  11/K/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

14022731   PMID: 12444318
 Monoclonal antibodies against Blo t 13, a recombinant allergen from
 Blomia tropicalis.
  Labrada Mayrel; Uyema Keiko; Sewer Minerva; Labrada Alexis; Gonzalez
Maritza; Caraballo Luis; Puerta Leonardo
  Department of Allergens, National Center of Bioproducts (BIOCEN), Havana,
Cuba.
  International archives of allergy and immunology (Switzerland)   Nov 2002
,  129  (3)  p212-8,  ISSN 1018-2438--Print   Journal Code: 9211652
  Publishing Model Print
```

Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

... against rBlot 13 from Escherichia coli and P. pastoris expression was compared. RESULTS: Two MAbs, 5G3 and 3G4 with IgG1 isotype, were generated. These MAbs specifically recognized the 16-kD band...

... molecular weight shown by rBlo t 13 on SDS-PAGE. In ELISA, the binding of 5G3 MAb to B. tropicalis and D. siboney extracts was inhibited by rBlo t 13. Both...
Descriptors: *Allergens--immunology--IM; *Antibodies, Monoclonal --immunology--IM; * Antibody Specificity--immunology--IM; *Carrier Proteins--immunology--IM; Animals; Binding Sites, Antibody --immunology --IM; Binding, Competitive--immunology--IM; Comparative Study; Cross Reactions--immunology--IM; Dose-Response Relationship...
Chemical Name: Allergens; Antibodies, Monoclonal; Binding Sites, Antibody ; Blo t 13 allergen; Carrier Proteins; Fatty Acid-Binding Proteins; Recombinant Proteins

11/K/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08989155 PMID: 1937498
The 200/220 kDa antigen recognized by monoclonal antibody (MAb) UJ127.11 on neural tissues and tumors is the human L1 adhesion molecule.
Patel K; Kiely F; Phimister E; Melino G; Rathjen F; Kemshead J T
Imperial Cancer Research Fund, Paediatric & Neuro-Oncology Group, Frenchay Hospital, Bristol, England.
Hybridoma (UNITED STATES) Aug 1991, 10 (4) p481-91, ISSN 0272-457X --Print Journal Code: 8202424
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

The 200/220 kDa antigen recognized by monoclonal antibody (MAb) UJ127.11 on neural tissues and tumors is the human L1 adhesion molecule.
...calculated molecular weight of between 200-220 kDa. Immunocytochemical studies with UJ127.11 and an antibody (5G3) recently utilized to isolate human L1 from brain indicate that both reagents have very similar ...

11/K/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07758369 PMID: 3136168
A human brain glycoprotein related to the mouse cell adhesion molecule L1.
Wolff J M; Frank R; Mujoo K; Spiro R C; Reisfeld R A; Rathjen F G
Max-Planck-Institut fur Entwicklungsbiologie, Tubingen, Federal Republic of Germany.
Journal of biological chemistry (UNITED STATES) Aug 25 1988, 263 (24) p11943-7, ISSN 0021-9258--Print Journal Code: 2985121R

Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

We have employed monoclonal antibody 5G3 , an antibody used to label human tumor cells of neural origin (Mujoo, K., Spiro, R.C., and...

... glycoprotein implicated predominantly in neurite-neurite interactions. On the basis of the following results the 5G3 antigen is considered to be the human homologue of mouse L1. In sodium dodecyl sulfate...
... their carbohydrate-depleted or undepleted components. In tryptic fingerprint analyses of the iodinated L1 and 5G3 components, 65% of the resolved peptides comigrated. Comparison of NH2-terminal amino acid sequences revealed a high degree of homology between human 5G3 and mouse L1, with 11 of 15 residues being identical. Furthermore, polyclonal antibodies to human 5G3 antigen were found to be cross-reactive with mouse L1 antigen and vice versa. All components of 5G3 and L1 antigens show considerable charge heterogeneity with partial overlapping of regions in isoelectric focusing...

11/K/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07071731 PMID: 3525541
Characterization of a unique glycoprotein antigen expressed on the surface of human neuroblastoma cells.
Mujoo K; Spiro R C; Reisfeld R A
Journal of biological chemistry (UNITED STATES) Aug 5 1986, 261 (22) p10299-305, ISSN 0021-9258--Print Journal Code: 2985121R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

... molecular probe to delineate chemical and biological characteristics of human neuroblastoma cells, a murine monoclonal antibody (Mab 5G3) was produced that is directed to a glycoprotein, preferentially expressed on the surface of such cells. This antibody is of IgG2a isotype, has an association constant of 8 X 10(9) M-1...

... observed with a variety of lymphoblastoid cell lines and normal fetal and adult tissues. Mab 5G3 specifically recognizes a neuroblastoma target glycoprotein antigen of 215 kDa that is derived from a...
... and expressed on the cell surface. A molecule of 200 kDa is detected by Mab 5G3 in spent culture medium of human neuroblastoma cells which is neither sulfated nor phosphorylated.

11/K/5 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

07386764 EMBASE No: 1998284395
Molecular analysis of polyreactive monoclonal antibodies from rheumatic carditis: Human anti-N-acetylglucosamine/anti-myosin antibody V region

genes
Adderson E.E.; Shikhman A.R.; Ward K.E.; Cunningham M.W.
Dr. M.W. Cunningham, Dept. of Microbiology and Immunology, Univ. of Oklahoma Hlth. Sci. Center, P.O. Box 26901, Oklahoma City, OK 73190 United States
AUTHOR EMAIL: madeleinecunningham@ouhsc.edu
Journal of Immunology (J. IMMUNOL.) (United States) 15 AUG 1998, 161/4 (2020-2031)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 67

Molecular analysis of polyreactive monoclonal antibodies from rheumatic carditis: Human anti-N-acetylglucosamine/anti-myosin antibody V region genes
DRUG DESCRIPTORS:
*monoclonal antibody --endogenous compound--ec; *myosin antibody --endogenous compound--ec; *n acetylglucosamine--endogenous compound--ec
MEDICAL DESCRIPTORS:
antibody detection; antigen recognition; epitope mapping; gene mutation; human; controlled study; article; priority journal
DRUG TERMS (UNCONTROLLED): monoclonal antibody 1 c8--endogenous compound --ec; monoclonal antibody 1 h9--endogenous compound--ec; monoclonal antibody 5g3 --endogenous compound--ec; monoclonal antibody 3 b6 --endogenous compound--ec
?

```
Set     Items   Description
S1        526   ((L1CAM) OR (L1 (N) CELL (N) ADHESION) AND ANTIBODY)
S2     971036   ((TUMOR OR TUMOUR OR CANCER OR NEOPLASIA OR CARCINOMA) AND
              (GROWTH OR PROLIFERATION))
S3         29   S1 AND S2
S4         13   RD S3  (unique items)
S5         50   (UJ127 AND ANTIBODY)
S6         19   RD S5  (unique items)
S7          3   (S6 AND (PROLIFERATION OR GROWTH))
S8          3   RD S7  (unique items)
S9         17   (5G3 AND ANTIBODY)
S10         0   (S9 AND (PROLIFERATION AND GROWTH))
S11         5   RD S9  (unique items)
?

S (L1-11A AND ANTIBODY)
               0  L1-11A
         1751004  ANTIBODY
     S12       0  (L1-11A AND ANTIBODY)
?

S (L1 (N) 11A) AND (ANTIBODY)
           65124  L1
            6795  11A
               4  L1(N)11A
         1751004  ANTIBODY
     S13       4  (L1 (N) 11A) AND (ANTIBODY)
?

RD S13
     S14       1  RD S13  (unique items)
```

```
?

TYPE S14/MEDIUM,K/1

  14/K/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

19910302   PMID: 16424028
 Efficient inhibition of intra-peritoneal tumor growth and dissemination
 of human ovarian carcinoma cells in nude mice by anti-L1-cell adhesion
 molecule monoclonal antibody treatment.
  Arlt Matthias J E; Novak-Hofer Ilse; Gast Daniela; Gschwend Verena;
Moldenhauer Gerhard; Grunberg Jurgen; Honer Michael; Schubiger P August;
Altevogt Peter; Kruger Achim
  Klinikum rechts der Isar der Technischen Universitat Munchen, Institut
fur Experimentelle Onkologie und Therapieforschung, Ismaninger Strasse 22,
D-81675 Munich, Germany.
  Cancer research (United States) Jan 15 2006, 66 (2) p936-43, ISSN
0008-5472--Print  Journal Code: 2984705R
  Publishing Model Print
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed

  ... up to -75%). This effect was associated with reduced proliferation
within the tumors. L1-directed antibody -based inhibition of peritoneal
growth and dissemination of human ovarian carcinoma cells represents
important proof...
?

S (CHCE7 AND ANTIBODY)
              94  CHCE7
         1751004  ANTIBODY
     S15      90  (CHCE7 AND ANTIBODY)
?

RD
     S16      43  RD  (unique items)
?

S (S16 AND (PROLIFERATION OR GROWTH))
              43  S16
          896535  PROLIFERATION
         4409277  GROWTH
     S17       5  (S16 AND (PROLIFERATION OR GROWTH))
?

RD
     S18       5  RD  (unique items)
?

TYPE S18/MEDIUM,K/1-5

  18/K/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

19910302   PMID: 16424028
```

Efficient inhibition of intra-peritoneal tumor growth and dissemination of human ovarian carcinoma cells in nude mice by anti-L1-cell adhesion molecule monoclonal antibody treatment.

Arlt Matthias J E; Novak-Hofer Ilse; Gast Daniela; Gschwend Verena; Moldenhauer Gerhard; Grunberg Jurgen; Honer Michael; Schubiger P August; Altevogt Peter; Kruger Achim

Klinikum rechts der Isar der Technischen Universitat Munchen, Institut fur Experimentelle Onkologie und Therapieforschung, Ismaninger Strasse 22, D-81675 Munich, Germany.

Cancer research (United States) Jan 15 2006, 66 (2) p936-43, ISSN 0008-5472--Print Journal Code: 2984705R

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Efficient inhibition of intra-peritoneal tumor growth and dissemination of human ovarian carcinoma cells in nude mice by anti-L1-cell adhesion molecule monoclonal antibody treatment.

The L1 cell adhesion molecule is implicated in the control of proliferation , migration, and invasion of several tumor cell types in vitro. Recently, L1 overexpression was found...

...potential target for ovarian cancer therapy, we investigated the effects of anti-L1 monoclonal antibodies (chCE7 and L1-11A) on proliferation and migration of L1-positive human SKOV3ip ovarian carcinoma cells in vitro and the therapeutic efficacy of L1-11A against i.p. SKOV3ip tumor growth in nude mice. In vitro, both anti-L1 antibodies efficiently inhibited the proliferation of SKOV3ip cells as well as other L1-expressing tumor cell lines (renal carcinoma, neuroblastoma...

... colon carcinoma). On two cell lines, hyper-cross-linking of L1-11A with a secondary antibody was necessary for significant inhibition of proliferation , indicating that cross-linking of L1 is required for the antiproliferative effect. L1-negative prostate carcinoma cells were not influenced by antibody treatment. Biweekly treatment of ovarian carcinoma-bearing mice with L1-11A led to a dose...

... to -63.5%) and ascites formation (up to -75%). This effect was associated with reduced proliferation within the tumors. L1-directed antibody -based inhibition of peritoneal growth and dissemination of human ovarian carcinoma cells represents important proof-of-principle for the development...

; Animals; Carcinoma--genetics--GE; Carcinoma--therapy--TH; Cell Movement; Cell Proliferation ; Disease Progression; Humans; Mice; Mice, Nude; Neural Cell Adhesion Molecule L1--physiology--PH; Ovarian Neoplasms ...

18/K/2 (Item 2 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

13185845 PMID: 11315605

A comparison of targeting of neuroblastoma with mIBG and anti L1-CAM antibody mAb chCE7: therapeutic efficacy in a neuroblastoma xenograft model and imaging of neuroblastoma patients.

Hoefnagel C A; Rutgers M; Buitenhuis C K; Smets L A; de Kraker J; Meli M; Carrel F; Amstutz H; Schubiger P A; Novak-Hofer I

Department of Nuclear Medicine, The Netherlands Cancer Institute, Amsterdam, The Netherlands.
European journal of nuclear medicine (Germany) Mar 2001, 28 (3) p359-68, ISSN 0340-6997--Print Journal Code: 7606882
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

A comparison of targeting of neuroblastoma with mIBG and anti L1-CAM antibody mAb chCE7 : therapeutic efficacy in a neuroblastoma xenograft model and imaging of neuroblastoma patients.

Iodine-131 labelled anti L1-CAM antibody mAb chCE7 was compared with the effective neuroblastoma-seeking agent 131I-labelled metaiodobenzylguanidine (MIBG) with regard to...

... uptake and provide a relatively low number of 6,300 binding sites/cell for mAb chCE7 . Tumours were treated with single injections of 131I-MIBG (110 MBq) and with 131I-labelled mAb chCE7 (17 MBq) and both agents showed antitumour activity. After therapy with 131I- chCE7 , the subcutaneous tumours nearly disappeared; treatment with 131I-MIBG was somewhat less effective, resulting in...

... of 9 days occurred with a radioactivity dose of 17 MBq of an irrelevant control antibody mAb 35, which does not bind to SK-N-SH cells, compared with a regrowth delay of 34 days with 131I-mAb chCE7 and of 24 days with 131I-MIBG. General toxicity appeared to be mild, as assessed by a transient, approximate 10% maximum decrease in body weight during the treatments. The superior growth inhibition achieved by 131I- chCE7 compared with 131I-MIBG can be explained by its prolonged retention in the tumours, due to slower normal tissue and plasma clearance. Cross-reaction of mAb chCE7 with L1-CAM present in normal human tissues was investigated by direct binding of radioiodinated...

... kidney sections. Seven patients with recurrent neuroblastoma were sequentially imaged with 131I-MIBG and 131I- chCE7 . The results underlined the heterogeneity of neuroblastoma and showed the two imaging modalities to be complementary. 131I- chCE7 scintigraphy may have clinical utility in detecting metastases which do not accumulate 131I-MIBG, and the antibody may hold potential for radioimmunotherapy, either by itself or in combination with 131I-MIBG.

18/K/3 (Item 1 from file: 159)
DIALOG(R)File 159:Cancerlit

02230344 PMID: 96604767
Construction of a single-chain F(V)-fragment of the chimeric anti-neuroblastoma antibody CHCE7 (Meeting abstract).
Amstutz; Carrel; Morgenthaler
Central Laboratory, Swiss Red Cross, 3000 Bern 22, Switzerland
Experientia 1995, 51, ISSN 0014-4754
Document Type: MEETING ABSTRACTS
Languages: ENGLISH
Main Citation Owner: NOTNLM
Record type: Completed

Construction of a single-chain F(V)-fragment of the chimeric

anti-neuroblastoma antibody CHCE7 (Meeting abstract).
The monoclonal antibody CE7 shows strong tumor selectivity by binding to a neuroblastoma-associated cell surface antigen. In...

...PCR. The CE7 V(H) and V(L) genes were expressed in the cassette and growth conditions for the secretion of the scF(V) were optimized. In JM101 there was no...

... screened for scF(V) expression. Variable amounts of expression levels were seen under the usual growth conditions. No secreted scF(V) was detectable. At lower growth temperatures, secreted scF(V) started to appear in the culture supernatant with an optimum at...

18/K/4 (Item 1 from file: 35)
DIALOG(R)File 35:Dissertation Abs Online

01659053 ORDER NO: AAD98-42322
ENGINEERING OF PROTEIN GLYCOSYLATION IN CHINESE HAMSTER OVARY CELLS (GLYCOFORMS)
Author: UMANA, PABLO
Degree: PH.D.
Year: 1998
Corporate Source/Institution: CALIFORNIA INSTITUTE OF TECHNOLOGY (0037)
Source: VOLUME 59/07-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 3589. 155 PAGES

...to maximize the proportion of beneficial glycoforms within the glycoform population. An anti-neuroblastoma monoclonal antibody (chCE7) was used as a model therapeutic glycoprotein, and the target glycoforms were those carrying bi...

...the experimental system, it was found that overexpression of GnTIII to high levels led to growth inhibition and was toxic to the cells.
A set of chCE7 mAb samples differing in their glycoform distributions was produced by controlling GnTIII expression in a...

...the ADCC activity of these samples showed an optimal range of GnTIII expression for maximal chCE7 in vitro biological activity. The activity correlated with the level of Fc-associated bisected, complex...

...by further engineering of the pathway could therefore be valuable.
The new optimized variants of chCE7 are promising candidate reagents for the treatment of neuroblastoma. The strategy presented here may also...

18/K/5 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

11638863 Genuine Article#: 678GW No. References: 15
Title: High-yield production of recombinant antibody fragments in HEK-293 cells using sodium butyrate
Author(s): Grunberg J (REPRINT) ; Knogler K; Waibel R; Novak-Hofer I
Corporate Source: Paul Scherrer Inst,Ctr Radiopharmaceut Sci,CH-5232 Villigen//Switzerland/ (REPRINT); ETH,PSI, USZ,CH-5232 Villigen//Switzerland/
Journal: BIOTECHNIQUES, 2003, V34, N5 (MAY), P968-+
ISSN: 0736-6205 Publication date: 20030500

Publisher: EATON PUBLISHING CO, 154 E. CENTRAL ST, NATICK, MA 01760 USA
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

Title: High-yield production of recombinant antibody fragments in HEK-293 cells using sodium butyrate

Abstract: To develop new recombinant monoclonal antibody fragments for therapy and imaging, it is indispensable to have a simple and easy procedure...

...drug methotrexate (for the DHFR system) can increase the production rate but decreases the specific growth rate of the cells. The production rate is not always stable over a long-term...
...in combination with an efficient screening method. Sodium butyrate can increase the espression of recombinant antibody fragments in the transfectomas up to 500 mug/4.2 x 10(7) cells/24...

...mug/mL culture medium. This strategy allows a rapid development of new recombinant mono-clonal antibody fragments and allows one to proceed rapidly to in vivo testing.
...Identifiers--EXPRESSION; NEUROBLASTOMA; CHCE7; GENE
?

Set	Items	Description
S1	526	((L1CAM) OR (L1 (N) CELL (N) ADHESION) AND ANTIBODY)
S2	971036	((TUMOR OR TUMOUR OR CANCER OR NEOPLASIA OR CARCINOMA) AND (GROWTH OR PROLIFERATION))
S3	29	S1 AND S2
S4	13	RD S3 (unique items)
S5	50	(UJ127 AND ANTIBODY)
S6	19	RD S5 (unique items)
S7	3	(S6 AND (PROLIFERATION OR GROWTH))
S8	3	RD S7 (unique items)
S9	17	(5G3 AND ANTIBODY)
S10	0	(S9 AND (PROLIFERATION AND GROWTH))
S11	5	RD S9 (unique items)
S12	0	(L1-11A AND ANTIBODY)
S13	4	(L1 (N) 11A) AND (ANTIBODY)
S14	1	RD S13 (unique items)
S15	90	(CHCE7 AND ANTIBODY)
S16	43	RD (unique items)
S17	5	(S16 AND (PROLIFERATION OR GROWTH))
S18	5	RD (unique items)

?

```
=> d his

     (FILE 'HOME' ENTERED AT 11:19:48 ON 19 MAY 2006)

     FILE 'CAPLUS, BIOENG, BIOTECHNO, BIOTECHDS, ESBIOBASE' ENTERED AT
     11:20:33 ON 19 MAY 2006
L1         23110 S  (((CELL (W) ADHESION) OR L1CAM) AND ANTIBODY)
L2          5521 S (L1 AND (PROLIFERATION OR GROWTH))
L3          2418 S (L2 AND (CANCER OR TUMOR OR TUMOUR OR NEOPLASIA OR CARCINOMA
L4         23103 S (L3 AND (L1 (W) ANTIGEN) OR (L1 (W) CELL (W) ADHESION))
L5             1 S (UJ127) AND L3
L6             5 S (UJ127) AND L4
L7             1 S L5 AND L6
L8             4 DUPLICATE REMOVE L6 (1 DUPLICATE REMOVED)
L9             1 S (5G3 AND L3)
L10            6 S (5G3 AND L4)
L11            4 DUPLICATE REMOVE L10 CAPLUS  (2 DUPLICATES REMOVED)
L12            4 DUPLICATE REMOVE L10 (2 DUPLICATES REMOVED)

=> s ((L1 (w) 11A) and antibody)
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L1 (W) 11A'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L2 (W) 11A'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L3 (W) 11A'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L4 (W) 11A'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L5 (W) 11A'
L13           24 ((L1 (W) 11A) AND ANTIBODY)

=> s l13 and l3
L14           13 L13 AND L3

=> s l13 and l4
L15           24 L13 AND L4

=> duplicate remove l14
DUPLICATE PREFERENCE IS 'CAPLUS, BIOENG, BIOTECHNO, ESBIOBASE'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L14
L16            10 DUPLICATE REMOVE L14 (3 DUPLICATES REMOVED)

=> duplicate remove for l15
'FOR' IS NOT VALID.  VALID FILE NAMES ARE 'CAPLUS, BIOENG, BIOTECHNO, ESBIOBASE'
You have entered a file name of duplicates to keep that is not
referenced by any of the L#s specified for this DUPLICATE command.
The file names of duplicates that can be kept are listed above.
Please enter one of these file names.
ENTER FILE NAMES OF DUPLICATES TO KEEP:caplus
PROCESSING COMPLETED FOR L15
L17            20 DUPLICATE REMOVE L15 CAPLUS  (4 DUPLICATES REMOVED)

=> duplicate remove l15
DUPLICATE PREFERENCE IS 'CAPLUS, BIOENG, BIOTECHNO, ESBIOBASE'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L15
L18            20 DUPLICATE REMOVE L15 (4 DUPLICATES REMOVED)

=> d his

     (FILE 'HOME' ENTERED AT 11:19:48 ON 19 MAY 2006)
```

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSPTALAB1643

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS	4	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS	5	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS	6	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS	7	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS	8	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS	9	MAR 22	EMBASE is now updated on a daily basis
NEWS	10	APR 03	New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS	11	APR 03	Bibliographic data updates resume; new IPC 8 fields and IPC thesaurus added in PCTFULL
NEWS	12	APR 04	STN AnaVist $500 visualization usage credit offered
NEWS	13	APR 12	LINSPEC, learning database for INSPEC, reloaded and enhanced
NEWS	14	APR 12	Improved structure highlighting in FQHIT and QHIT display in MARPAT
NEWS	15	APR 12	Derwent World Patents Index to be reloaded and enhanced during second quarter; strategies may be affected
NEWS	16	MAY 10	CA/CAplus enhanced with 1900-1906 U.S. patent records
NEWS	17	MAY 11	KOREAPAT updates resume

NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
http://download.cas.org/express/v8.0-Discover/

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8
NEWS X25	X.25 communication option no longer available after June 2006

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* *

COMPLETE THE STN SURVEY - APRIL 27 THROUGH MAY 31

Dear valued STN customer,

In an effort to enhance your experience with STN, we would like to better understand what you find useful. Please take approximately 5 minutes to complete a web survey.

If you provide us with your name, login ID, and e-mail address, you will be entered in a drawing to win a free iPod(R). Your responses will be kept confidential and will help us make future improvements to STN.

Take survey: http://www.zoomerang.com/survey.zgi?p=WEB2259HNKWTUW

Thank you in advance for your participation.

```
* * * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 11:19:48 ON 19 MAY 2006

=> ile caplus, bioeng, biotechno, biotechds, esbiobase
ILE IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> file  caplus, bioeng, biotechno, biotechds, esbiobase
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'CAPLUS' ENTERED AT 11:20:33 ON 19 MAY 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'BIOENG' ENTERED AT 11:20:33 ON 19 MAY 2006
COPYRIGHT (C) 2006 Cambridge Scientific Abstracts (CSA)

FILE 'BIOTECHNO' ENTERED AT 11:20:33 ON 19 MAY 2006
COPYRIGHT (C) 2006 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'BIOTECHDS' ENTERED AT 11:20:33 ON 19 MAY 2006
COPYRIGHT (C) 2006 THE THOMSON CORPORATION

FILE 'ESBIOBASE' ENTERED AT 11:20:33 ON 19 MAY 2006
COPYRIGHT (C) 2006 Elsevier Science B.V., Amsterdam. All rights reserved.

=> s (((L1 (w) cell (w) adhesion) or L1CAM) and antibody)
L1 NOT FOUND
The L-number entered could not be found.  To see the definition
of L-numbers, enter DISPLAY HISTORY at an arrow prompt (=>).

=> s  (((cell (w) adhesion) or L1CAM) and antibody)
   4 FILES SEARCHED...
L1        23110 (((CELL (W) ADHESION) OR L1CAM) AND ANTIBODY)

=> s (L1 and (proliferation or growth))
L2         5521 (L1 AND (PROLIFERATION OR GROWTH))

=> s (L2 and (cancer or tumor or tumour or neoplasia or carcinoma))
L3         2418 (L2 AND (CANCER OR TUMOR OR TUMOUR OR NEOPLASIA OR CARCINOMA))

=> s (L3 and (L1 (w) antigen) or (L1 (w) cell (w) adhesion))
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L1 (W) ANTIGEN'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
```

```
FIELD CODE - 'AND' OPERATOR ASSUMED 'L1 (W) CELL'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'CELL (W) ADHESION'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L2 (W) ANTIGEN'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L2 (W) CELL'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'CELL (W) ADHESION'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L3 (W) ANTIGEN'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L3 (W) CELL'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'CELL (W) ADHESION'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L4 (W) ANTIGEN'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L4 (W) CELL'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'CELL (W) ADHESION'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L5 (W) ANTIGEN'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L5 (W) CELL'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'CELL (W) ADHESION'
L4       23103 (L3 AND (L1 (W) ANTIGEN) OR (L1 (W) CELL (W) ADHESION))

=> d his

     (FILE 'HOME' ENTERED AT 11:19:48 ON 19 MAY 2006)

     FILE 'CAPLUS, BIOENG, BIOTECHNO, BIOTECHDS, ESBIOBASE' ENTERED AT
     11:20:33 ON 19 MAY 2006
L1        23110 S  (((CELL (W) ADHESION) OR L1CAM) AND ANTIBODY)
L2         5521 S (L1 AND (PROLIFERATION OR GROWTH))
L3         2418 S (L2 AND (CANCER OR TUMOR OR TUMOUR OR NEOPLASIA OR CARCINOMA
L4        23103 S (L3 AND (L1 (W) ANTIGEN) OR (L1 (W) CELL (W) ADHESION))

=> s (UJ127) and L3
L5            1 (UJ127) AND L3

=> s (UJ127) and L4
L6            5 (UJ127) AND L4

=> s L5 and L6
L7            1 L5 AND L6

=> d l5 bib abs 1

L5   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2006 ACS on STN
AN   2004:368887  CAPLUS
DN   140:373908
TI   Antibody-mediated induction of tumor cell death
IN   Primiano, Thomas; Roninson, Igor B.
PA   The Board of Trustees of the University of Illinois, USA
SO   PCT Int. Appl., 22 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.           KIND   DATE        APPLICATION NO.        DATE
     ---------------      ----   --------    --------------------   --------
```

PI WO 2004037198 A2 20040506 WO 2003-US33712 20031023
WO 2004037198 A3 20041202
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
AU 2003286645 A1 20040513 AU 2003-286645 20031023
US 2004115206 A1 20040617 US 2003-692303 20031023
PRAI US 2002-420963P P 20021024
US 2003-483684P P 20030630
US 2003-485590P P 20030708
WO 2003-US33712 W 20031023
AB The disclosed invention provides methods and reagents for inducing cell death in **tumor** cells. The invention provides said reagents relating to inducing **tumor** cell death that are **antibodies** to a specific target, neural **cell adhesion** mol. **L1CAM**, and methods for using said **antibodies** for inducing cell death. Pharmaceutical compns. of the **L1CAM antibodies** for use in the practice of the methods of the invention are also disclosed. The example presents the effects of 2 anti-**L1CAM** monoclonal **antibodies** (**UJ127** and 5G3) on **growth** of 4 human **tumor** cell lines (2 breast **carcinoma**, cervical **carcinoma**, and colon **carcinoma**) and 4 normal human cell lines (normal human fibroblasts and 3 human mammary epithelial cell lines). The addition of either **UJ127** or 5G3 **antibody** to the cell cutlure media resulted in 3-6-fold decrease in the number of **tumor** cells, with little or no **growth** inhibition in any of the normal cells. Similar results were observed using a rabbit polyclonal anti-**L1CAM** antiserum.

=> d his

(FILE 'HOME' ENTERED AT 11:19:48 ON 19 MAY 2006)

FILE 'CAPLUS, BIOENG, BIOTECHNO, BIOTECHDS, ESBIOBASE' ENTERED AT 11:20:33 ON 19 MAY 2006
L1 23110 S (((CELL (W) ADHESION) OR L1CAM) AND ANTIBODY)
L2 5521 S (L1 AND (PROLIFERATION OR GROWTH))
L3 2418 S (L2 AND (CANCER OR TUMOR OR TUMOUR OR NEOPLASIA OR CARCINOMA
L4 23103 S (L3 AND (L1 (W) ANTIGEN) OR (L1 (W) CELL (W) ADHESION))
L5 1 S (UJ127) AND L3
L6 5 S (UJ127) AND L4
L7 1 S L5 AND L6

=> duplicate remove l6
DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHNO'
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETED FOR L6
L8 4 DUPLICATE REMOVE L6 (1 DUPLICATE REMOVED)

=> d l8 bib abs 1-4

L8 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2006:171817 CAPLUS
DN 144:271896
TI L1 is a potential marker for poorly-differentiated pancreatic

neuroendocrine carcinoma
AU Kaifi, Jussuf T.; Zinnkann, Ulrich; Yekebas, Emre F.; Schurr, Paulus G.; Reichelt, Uta; Wachowiak, Robin; Fiegel, Henning C.; Petri, Susann; Schachner, Melitta; Izbicki, Jakob R.
CS Department of General, Visceral and Thoracic Surgery, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
SO World Journal of Gastroenterology (2006), 12(1), 94-98
CODEN: WJGAF2; ISSN: 1007-9327
PB World Journal of Gastroenterology
DT Journal
LA English
AB AIM: To determine the expression of L1 in pancreatic neuroendocrine tumor and to correlate it with WHO classification of this tumor. METHODS: We retrospectively analyzed L1 expression in 63 cases of pancreatic neuroendocrine tumor by immunohistochem. on paraffin sections of primary tumors or metastases. Staining was performed by peroxidase technique with monoclonal **antibody UJ127**.11 against human L1. All tumors were classified according to WHO classification as well-differentiated neuroendocrine tumors and carcinomas or poorly-differentiated neuroendocrine carcinomas. RESULTS: L1 was detected in 5 (7.9%) of 63 pancreatic neuroendocrine tumors. Four (44.4%) of 9 poorly-differentiated carcinomas expressed L1. In contrast, only 1 (1.9%) of 54 well-differentiated tumors or carcinomas was pos. for L1. No expression was found in Langerhans islet **cells** of normal pancreatic tissue. Cross table anal. showed a significant association between L1 expression and classification of neuroendocrine tumors of the pancreas ($P<0.01$). CONCLUSION: L1 is specifically expressed in poorly-differentiated pancreatic neuroendocrine carcinomas that are known to have the worst prognosis. L1 might be a marker for risk prediction of patients diagnosed with pancreatic neuroendocrine carcinomas.
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2004:368887 CAPLUS
DN 140:373908
TI **Antibody**-mediated induction of tumor **cell** death
IN Primiano, Thomas; Roninson, Igor B.
PA The Board of Trustees of the University of Illinois, USA
SO PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004037198	A2	20040506	WO 2003-US33712	20031023
	WO 2004037198	A3	20041202		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003286645	A1	20040513	AU 2003-286645	20031023
	US 2004115206	A1	20040617	US 2003-692303	20031023
PRAI	US 2002-420963P	P	20021024		
	US 2003-483684P	P	20030630		
	US 2003-485590P	P	20030708		
	WO 2003-US33712	W	20031023		

AB The disclosed invention provides methods and reagents for inducing **cell** death in tumor **cells**. The invention provides said reagents relating to inducing tumor **cell** death that are **antibodies** to a specific target, neural **cell adhesion** mol. **L1CAM**, and methods for using said **antibodies** for inducing **cell** death. Pharmaceutical compns. of the **L1CAM antibodies** for use in the practice of the methods of the invention are also disclosed. The example presents the effects of 2 anti-**L1CAM** monoclonal **antibodies** (**UJ127** and 5G3) on growth of 4 human tumor **cell** lines (2 breast carcinoma, cervical carcinoma, and colon carcinoma) and 4 normal human **cell** lines (normal human fibroblasts and 3 human mammary epithelial **cell** lines). The addition of either **UJ127** or 5G3 **antibody** to the **cell** cutlure media resulted in 3-6-fold decrease in the number of tumor **cells**, with little or no growth inhibition in any of the normal **cells**. Similar results were observed using a rabbit polyclonal anti-**L1CAM** antiserum.

L8 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1
AN 1991:676786 CAPLUS
DN 115:276786
TI The 200/220 kDa antigen recognized by monoclonal **antibody** (MAb) **UJ127**.11 on neural tissues and tumors is the human L1 **adhesion** molecule
AU Patel, K.; Kiely, F.; Phimister, E.; Melino, G.; Rathjen, F.; Kemshead, J. T.
CS Imp. Cancer Res. Fund, Frenchay Hosp., Bristol, BS16 1LE, UK
SO Hybridoma (1991), 10(4), 481-91
CODEN: HYBRDY; ISSN: 0272-457X
DT Journal
LA English
AB MAb **UJ127**.11, raised against 16-wk human fetal brain, recognizes an antigen present primarily on normal and tumor tissues derived from the neuroectoderm. The antigen was previously identified as a 220/240 kDa **cell** surface glycoprotein, as determined by immunopptn. studies. The 220/240 kDa antigen is the human L1 **cell adhesion** mol. Western blot anal. confirmed the calculated mol. weight of 200-220 kDa. Immunocytochem. studies with **UJ127**.11 and an **antibody** (5G3) recently utilized to isolate human L1 from brain indicate that both reagents have very similar binding profiles. The binding of radiolabeled **UJ127**.11 to its target antigen can be blocked by the addition of a rabbit anti-human L1 antiserum. Sequential immunopptn. and Western blot anal. show that **UJ127**.11 and the rabbit anti-human L1 antiserum recognize identical proteins.

L8 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1991:574173 CAPLUS
DN 115:174173
TI Retinoic acid and α-difluoromethylornithine induce different expression of neural-specific **cell adhesion** molecules in differentiating neuroblastoma **cells**
AU Melino, G.; Piacentini, M.; Patel, K.; Annicchiarico-Petruzzelli, M.; Piredda, L.; Kemshead, J. T.
CS Dep. Exp. Med., Tor Vergata Univ., Rome, 00173, Italy
SO Progress in Clinical and Biological Research (1991), 366(Adv. Neuroblastoma Res. 3), 283-91
CODEN: PCBRD2; ISSN: 0361-7742
DT Journal
LA English
AB Human neuroblastoma **cells** SK-N-BE(2) can be induced to (RA) or a schwannian/glial phenotype by α-difluoromethylornithine (DFMO), producing differential binding of 14 **antibodies** (MAbs). RA induced the expression of the neural **cell adhesion**

mol., NCAM (also confirmed by northern blot); whereas DFMO enhanced the binding of the MAbs UJ181.4 and **UJ127**.11 which recognize an identical protein doublet of 220-240 kDa, thought to be the L1 protein(s). The data presented demonstrate that neuroblastoma **cells** differentiate toward sep. phenotypes associated with a specific induction of two different **adhesion** mols., NCAM on neuronal **cells** and L1 on schwannian/glial **cells**.

```
=> d his

     (FILE 'HOME' ENTERED AT 11:19:48 ON 19 MAY 2006)

     FILE 'CAPLUS, BIOENG, BIOTECHNO, BIOTECHDS, ESBIOBASE' ENTERED AT
     11:20:33 ON 19 MAY 2006
L1        23110 S  (((CELL (W) ADHESION) OR L1CAM) AND ANTIBODY)
L2         5521 S (L1 AND (PROLIFERATION OR GROWTH))
L3         2418 S (L2 AND (CANCER OR TUMOR OR TUMOUR OR NEOPLASIA OR CARCINOMA
L4        23103 S (L3 AND (L1 (W) ANTIGEN) OR (L1 (W) CELL (W) ADHESION))
L5            1 S (UJ127) AND L3
L6            5 S (UJ127) AND L4
L7            1 S L5 AND L6
L8            4 DUPLICATE REMOVE L6 (1 DUPLICATE REMOVED)

=> s (5G3 and L3)
L9            1 (5G3 AND L3)

=> s (5G3 and L4)
L10           6 (5G3 AND L4)

=> d l9 bib abs 1

L9   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2006 ACS on STN
AN   2004:368887  CAPLUS
DN   140:373908
TI   Antibody-mediated induction of tumor cell death
IN   Primiano, Thomas; Roninson, Igor B.
PA   The Board of Trustees of the University of Illinois, USA
SO   PCT Int. Appl., 22 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     --------------      ----   --------    --------------------    --------
PI   WO 2004037198        A2     20040506    WO 2003-US33712         20031023
     WO 2004037198        A3     20041202
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     AU 2003286645        A1     20040513    AU 2003-286645          20031023
     US 2004115206        A1     20040617    US 2003-692303          20031023
PRAI US 2002-420963P      P      20021024
     US 2003-483684P      P      20030630
     US 2003-485590P      P      20030708
     WO 2003-US33712      W      20031023
AB   The disclosed invention provides methods and reagents for inducing cell
```

death in **tumor** cells. The invention provides said reagents relating to inducing **tumor** cell death that are **antibodies** to a specific target, neural **cell adhesion** mol. **L1CAM**, and methods for using said **antibodies** for inducing cell death. Pharmaceutical compns. of the **L1CAM antibodies** for use in the practice of the methods of the invention are also disclosed. The example presents the effects of 2 anti-**L1CAM** monoclonal **antibodies** (UJ127 and **5G3**) on **growth** of 4 human **tumor** cell lines (2 breast **carcinoma**, cervical **carcinoma**, and colon **carcinoma**) and 4 normal human cell lines (normal human fibroblasts and 3 human mammary epithelial cell lines). The addition of either UJ127 or **5G3 antibody** to the cell cutlure media resulted in 3-6-fold decrease in the number of **tumor** cells, with little or no **growth** inhibition in any of the normal cells. Similar results were observed using a rabbit polyclonal anti-**L1CAM** antiserum.

```
=> d his

     (FILE 'HOME' ENTERED AT 11:19:48 ON 19 MAY 2006)

     FILE 'CAPLUS, BIOENG, BIOTECHNO, BIOTECHDS, ESBIOBASE' ENTERED AT
     11:20:33 ON 19 MAY 2006
L1         23110 S  (((CELL (W) ADHESION) OR L1CAM) AND ANTIBODY)
L2          5521 S (L1 AND (PROLIFERATION OR GROWTH))
L3          2418 S (L2 AND (CANCER OR TUMOR OR TUMOUR OR NEOPLASIA OR CARCINOMA
L4         23103 S (L3 AND (L1 (W) ANTIGEN) OR (L1 (W) CELL (W) ADHESION))
L5             1 S (UJ127) AND L3
L6             5 S (UJ127) AND L4
L7             1 S L5 AND L6
L8             4 DUPLICATE REMOVE L6 (1 DUPLICATE REMOVED)
L9             1 S (5G3 AND L3)
L10            6 S (5G3 AND L4)

=> duplicate remove L10
DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHNO'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):y
ENTER FILE NAMES OF DUPLICATES TO KEEP:n
'N' IS NOT VALID.  VALID FILE NAMES ARE 'CAPLUS, BIOTECHNO'
You have entered a file name of duplicates to keep that is not
referenced by any of the L#s specified for this DUPLICATE command.
The file names of duplicates that can be kept are listed above.
Please enter one of these file names.
ENTER FILE NAMES OF DUPLICATES TO KEEP: caplus
PROCESSING COMPLETED FOR L10
L11            4 DUPLICATE REMOVE L10 CAPLUS  (2 DUPLICATES REMOVED)

=> duplicate remove l10
DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHNO'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L10
L12            4 DUPLICATE REMOVE L10 (2 DUPLICATES REMOVED)

=> d his

     (FILE 'HOME' ENTERED AT 11:19:48 ON 19 MAY 2006)

     FILE 'CAPLUS, BIOENG, BIOTECHNO, BIOTECHDS, ESBIOBASE' ENTERED AT
     11:20:33 ON 19 MAY 2006
L1         23110 S  (((CELL (W) ADHESION) OR L1CAM) AND ANTIBODY)
L2          5521 S (L1 AND (PROLIFERATION OR GROWTH))
L3          2418 S (L2 AND (CANCER OR TUMOR OR TUMOUR OR NEOPLASIA OR CARCINOMA
L4         23103 S (L3 AND (L1 (W) ANTIGEN) OR (L1 (W) CELL (W) ADHESION))
```

```
L5              1 S (UJ127) AND L3
L6              5 S (UJ127) AND L4
L7              1 S L5 AND L6
L8              4 DUPLICATE REMOVE L6 (1 DUPLICATE REMOVED)
L9              1 S (5G3 AND L3)
L10             6 S (5G3 AND L4)
L11             4 DUPLICATE REMOVE L10 CAPLUS  (2 DUPLICATES REMOVED)
L12             4 DUPLICATE REMOVE L10 (2 DUPLICATES REMOVED)

=> d l12 bib abs

L12  ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2006 ACS on STN
AN   2004:368887  CAPLUS
DN   140:373908
TI   Antibody-mediated induction of tumor cell death
IN   Primiano, Thomas; Roninson, Igor B.
PA   The Board of Trustees of the University of Illinois, USA
SO   PCT Int. Appl., 22 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     ---------------      ----   --------    --------------------    --------
PI   WO 2004037198         A2    20040506    WO 2003-US33712         20031023
     WO 2004037198         A3    20041202
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     AU 2003286645         A1    20040513    AU 2003-286645          20031023
     US 2004115206         A1    20040617    US 2003-692303          20031023
PRAI US 2002-420963P       P     20021024
     US 2003-483684P       P     20030630
     US 2003-485590P       P     20030708
     WO 2003-US33712       W     20031023
AB   The disclosed invention provides methods and reagents for inducing
     cell death in tumor cells.  The invention provides said
     reagents relating to inducing tumor cell death that are
     antibodies to a specific target, neural cell
     adhesion mol. L1CAM, and methods for using said
     antibodies for inducing cell death.  Pharmaceutical
     compns. of the L1CAM antibodies for use in the
     practice of the methods of the invention are also disclosed.  The example
     presents the effects of 2 anti-L1CAM monoclonal
     antibodies (UJ127 and 5G3) on growth of 4 human tumor
     cell lines (2 breast carcinoma, cervical carcinoma, and colon
     carcinoma) and 4 normal human cell lines (normal human
     fibroblasts and 3 human mammary epithelial cell lines).  The
     addition of either UJ127 or 5G3 antibody to the
     cell cutlure media resulted in 3-6-fold decrease in the number of
     tumor cells, with little or no growth inhibition in any of the
     normal cells.  Similar results were observed using a rabbit
     polyclonal anti-L1CAM antiserum.


=> d his
```

(FILE 'HOME' ENTERED AT 11:19:48 ON 19 MAY 2006)

FILE 'CAPLUS, BIOENG, BIOTECHNO, BIOTECHDS, ESBIOBASE' ENTERED AT 11:20:33 ON 19 MAY 2006

```
L1        23110 S  (((CELL (W) ADHESION) OR L1CAM) AND ANTIBODY)
L2         5521 S (L1 AND (PROLIFERATION OR GROWTH))
L3         2418 S (L2 AND (CANCER OR TUMOR OR TUMOUR OR NEOPLASIA OR CARCINOMA
L4        23103 S (L3 AND (L1 (W) ANTIGEN) OR (L1 (W) CELL (W) ADHESION))
L5            1 S (UJ127) AND L3
L6            5 S (UJ127) AND L4
L7            1 S L5 AND L6
L8            4 DUPLICATE REMOVE L6 (1 DUPLICATE REMOVED)
L9            1 S (5G3 AND L3)
L10           6 S (5G3 AND L4)
L11           4 DUPLICATE REMOVE L10 CAPLUS  (2 DUPLICATES REMOVED)
L12           4 DUPLICATE REMOVE L10 (2 DUPLICATES REMOVED)
```

=> d l12 bib abs 2-4

L12 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2002:275835 CAPLUS
DN 136:273193
TI Methods and compositions for modulating T **cell** activation and uses thereof
IN Montgomery, Anthony; Balaian, Larissa
PA The Scripps Research Institute, USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002028440	A1	20020411	WO 2001-US30864	20011002
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2423436	AA	20020411	CA 2001-2423436	20011002
	AU 2002013001	A5	20020415	AU 2002-13001	20011002
	EP 1328298	A1	20030723	EP 2001-981352	20011002
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2004531457	T2	20041014	JP 2002-532264	20011002
	US 2003040477	A1	20030227	US 2002-130087	20020923
PRAI	US 2000-237555P	P	20001002		
	WO 2001-US30864	W	20011002		

AB This invention relates generally to the field of immunol. or neuroimmunol. In particular, the invention provides a method for reducing or inhibiting T **cell** activation, which method comprises administering an effective amount of an antagonist of NCAM L1 to a mammal, wherein reduction or inhibition of T **cell** activation is desirable, thereby reducing or inhibiting T **cell** activation in said mammal. Combinations and combinatorial methods for modulating T **cell** activation are further provided. The invention also provides a method for potentiating T **cell** activation, which method comprises administering an effective amount of a multimerized neural **cell adhesion** mol. L1 (NCAM L1), or a functional derivative or fragment thereof, or a nucleic acid encoding said L1 or functional derivative or fragment thereof, or an agent

that enhances production and/or costimulatory function of said L1 to a mammal, wherein T **cell** activation is desirable, thereby potentiating T **cell** activation in said mammal.

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1
AN 1991:676786 CAPLUS
DN 115:276786
TI The 200/220 kDa antigen recognized by monoclonal **antibody** (MAb) UJ127.11 on neural tissues and tumors is the human L1 **adhesion** molecule
AU Patel, K.; Kiely, F.; Phimister, E.; Melino, G.; Rathjen, F.; Kemshead, J. T.
CS Imp. Cancer Res. Fund, Frenchay Hosp., Bristol, BS16 1LE, UK
SO Hybridoma (1991), 10(4), 481-91
CODEN: HYBRDY; ISSN: 0272-457X
DT Journal
LA English
AB MAb UJ127.11, raised against 16-wk human fetal brain, recognizes an antigen present primarily on normal and tumor tissues derived from the neuroectoderm. The antigen was previously identified as a 220/240 kDa **cell** surface glycoprotein, as determined by immunopptn. studies. The 220/240 kDa antigen is the human L1 **cell adhesion** mol. Western blot anal. confirmed the calculated mol. weight of 200-220 kDa. Immunocytochem. studies with UJ127.11 and an **antibody** (**5G3**) recently utilized to isolate human L1 from brain indicate that both reagents have very similar binding profiles. The binding of radiolabeled UJ127.11 to its target antigen can be blocked by the addition of a rabbit anti-human L1 antiserum. Sequential immunopptn. and Western blot anal. show that UJ127.11 and the rabbit anti-human L1 antiserum recognize identical proteins.

L12 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2
AN 1988:526349 CAPLUS
DN 109:126349
TI A human brain glycoprotein related to the mouse **cell adhesion** molecule L1
AU Wolff, J. Michael; Frank, Rainer; Mujoo, Kalpana; Spiro, Robert C.; Reisfeld, Ralph A.; Rathjen, Fritz G.
CS Max-Planck-Inst. Entwicklungsbiol., Tuebingen, D-7400, Fed. Rep. Ger.
SO Journal of Biological Chemistry (1988), 263(24), 11943-7
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
AB Monoclonal **antibody 5G3**, an **antibody** used to label human tumor **cells** of neural origin, was used to isolate and characterize a large glycoprotein from normal adult human brain. This protein was compared to mouse L1, a neural **cell** surface glycoprotein implicated predominantly in neurite-neurite interactions. On the basis of the following results the **5G3** antigen is considered to be the human homolog of mouse L1. In SDS-PAGE, both proteins share similar mol. masses of their carbohydrate-depleted or undepleted components. In tryptic fingerprint analyses of the iodinated L1 and **5G3** components, 65% of the resolved peptides comigrated. Comparison of N-terminal amino acid sequences revealed a high degree of homol. between human **5G3** and mouse L1, with 11 of 15 residues being identical. Furthermore, polyclonal **antibodies** to human **5G3** antigen were cross-reactive with mouse L1 antigen and vice versa. All components of **5G3** and L1 antigens showed considerable charge heterogeneity with partial overlapping of regions in isoelec. focusing followed by SDS-PAGE. These findings provide a basis for studying the role of the human L1 homolog in human diseases.

```
     FILE 'CAPLUS, BIOENG, BIOTECHNO, BIOTECHDS, ESBIOBASE' ENTERED AT
     11:20:33 ON 19 MAY 2006
L1        23110 S  (((CELL (W) ADHESION) OR L1CAM) AND ANTIBODY)
L2         5521 S (L1 AND (PROLIFERATION OR GROWTH))
L3         2418 S (L2 AND (CANCER OR TUMOR OR TUMOUR OR NEOPLASIA OR CARCINOMA
L4        23103 S (L3 AND (L1 (W) ANTIGEN) OR (L1 (W) CELL (W) ADHESION))
L5            1 S (UJ127) AND L3
L6            5 S (UJ127) AND L4
L7            1 S L5 AND L6
L8            4 DUPLICATE REMOVE L6 (1 DUPLICATE REMOVED)
L9            1 S (5G3 AND L3)
L10           6 S (5G3 AND L4)
L11           4 DUPLICATE REMOVE L10 CAPLUS  (2 DUPLICATES REMOVED)
L12           4 DUPLICATE REMOVE L10 (2 DUPLICATES REMOVED)
L13          24 S ((L1 (W) 11A) AND ANTIBODY)
L14          13 S L13 AND L3
L15          24 S L13 AND L4
L16          10 DUPLICATE REMOVE L14 (3 DUPLICATES REMOVED)
L17          20 DUPLICATE REMOVE L15 CAPLUS  (4 DUPLICATES REMOVED)
L18          20 DUPLICATE REMOVE L15 (4 DUPLICATES REMOVED)

=> d l16 bib abs 1-10

L16  ANSWER 1 OF 10  CAPLUS  COPYRIGHT 2006 ACS on STN
AN   2006:29606  CAPLUS
DN   144:121754
TI   Gene expression profile for predicting activity of compounds that interact
     with and/or modulate protein tyrosine kinases and/or protein tyrosine
     pathways in lung cancer cells
IN   Huang, Fei; Reeves, Karen A.; Han, Xia; Fairchild, Craig R.; Shaw, Peter
PA   Bristol-Myers Squibb Company, USA
SO   PCT Int. Appl., 130 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.          DATE
     ---------------     ----   --------    --------------------     --------
PI   WO 2006005035        A2    20060112    WO 2005-US23687          20050629
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ,
             LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA,
             NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK,
             SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU,
             ZA, ZM, ZW
         RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
             IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF,
             CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM,
             KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG,
             KZ, MD, RU, TJ, TM
     US 2006019284         A1    20060126    US 2005-169041           20050628
PRAI US 2004-584405P       P     20040630
AB   The present invention describes polynucleotides that have been discovered
     to correlate to the relative intrinsic sensitivity or resistance of cells,
     e.g., lung cell lines, to treatment with compds. that interact with and
     modulate, e.g., inhibit, protein tyrosine kinases, such as, for example,
     members of the Src family of tyrosine kinases, e.g., Src, Fgr, Fyn, Yes,
     Blk, Hck, Lck and Lyn, as well as other protein tyrosine kinases,
     including, Bcr-abl, Jak, PDGFR, c-kit and Ephr.  These polynucleotides
     have been shown, through a weighted voting cross validation program, to
     have utility in predicting the resistance and sensitivity of lung cell
     lines to the compds.  The expression level of some polynucleotides is
```

regulated by treatment with a particular protein tyrosine kinase inhibitor compound, thus indicating that these polynucleotides are involved in the protein tyrosine kinase signal transduction pathway, e.g., Src tyrosine kinase. The Affymetrix human HG-U133 GeneChip set of over 44,792 probe sets was used to identify 129 polynucleotides that are highly correlated with a resistance/sensitivity phenotype classification of 23 lung cell lines subjected to treatment with the protein tyrosine kinase inhibitor compound BMS-A. Of the 129 predictor polynucleotides, 81 polynucleotides highly expressed in the cell lines were classified as sensitive to BMS-A, while 48 polynucleotides highly expressed in the cell lines were classified as resistant to BMS-A. Such polynucleotides, whose expression levels correlate highly with drug sensitivity or resistance and which are modulated by treatment with the compds., comprise polynucleotide predictor or marker sets useful in methods of predicting drug response, and as prognostic or diagnostic indicators in disease management, particularly in those disease areas, e.g., lung **cancer**, in which signaling through the protein tyrosine kinase pathway, such as the Src tyrosine kinase pathway, is involved with the disease process.

L16 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2006:338002 CAPLUS
DN 144:383459
TI Screening parkinson's disease therapeutics based on genes differentially expressed in A9 dopaminergic neurons
IN Isacson, Ole
PA USA
SO U.S. Pat. Appl. Publ., 35 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2006078890	A1	20060413	US 2004-962080	20041008
WO 2006042137	A2	20060420	WO 2005-US36208	20051007

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI US 2004-962080 A 20041008
AB The present invention features methods of identifying compds. useful for the treatment and prevention of Parkinson's disease (PD). The invention is based on our discovery of numerous genes that are differentially expressed in A9 dopaminergic neurons, which undergo a disproportionately high level of cell death in PD, compared to A10 dopaminergic neurons, which are relatively spared. Compds. that reduce or prevent neurodegeneration caused by PD can be identified using screening methods that employ the genes and/or polypeptides that are differentially expressed in neurodegeneration-sensitive (A9) and neurodegeneration-resistant (A10) cells. Screening methods that make use of a plurality of such genes and polypeptides allow for the identification of agents associated with an improved ability to specifically and effectively treat and prevent neurodegeneration. Microarray anal. was performed to investigate the mol. differences between dopaminergic neurons located in the A9 and A10 midbrain regions. The differences that distinguished these two neuronal populations illustrated that only a small number of genes were differentially

expressed. Forty-six genes had greater than 2.0-fold elevation of mRNA levels in A9 compared to A10 DA neurons, and 199 genes, greater than 1.5-fold [false discovery rate (FDR)<5 %]. Sixty-one genes had greater than 2.0-fold elevation of mRNA level in A10 compared to A9 DA neurons and 163 genes, greater than 1.5 fold (FDR<5 %) (Tables 4 and 5).

L16 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1
AN 2006:55798 CAPLUS
DN 144:127146
TI Efficient Inhibition of Intra-Peritoneal **Tumor Growth** and Dissemination of Human Ovarian **Carcinoma** Cells in Nude Mice by Anti-L1-**Cell Adhesion** Molecule Monoclonal **Antibody** Treatment
AU Arlt, Matthias J. E.; Novak-Hofer, Ilse; Gast, Daniela; Gschwend, Verena; Moldenhauer, Gerhard; Gruenberg, Juergen; Honer, Michael; Schubiger, P. August; Altevogt, Peter; Krueger, Achim
CS Klinikum rechts der Isar, Technischen Universitaet Muenchen, Munich, Germany
SO Cancer Research (2006), 66(2), 936-943
CODEN: CNREA8; ISSN: 0008-5472
PB American Association for Cancer Research
DT Journal
LA English
AB The L1 **cell adhesion** mol. is implicated in the control of **proliferation**, migration, and invasion of several **tumor** cell types in vitro. Recently, L1 overexpression was found to correlate with **tumor** progression of ovarian **carcinoma** , one of the most common causes of **cancer**-related deaths in gynecol. malignant diseases. To evaluate L1 as a potential target for ovarian **cancer** therapy, the authors investigated the effects of anti-L1 monoclonal **antibodies** (chCE7 and L1-**11A**) on **proliferation** and migration of L1-pos. human SKOV3i.p. ovarian **carcinoma** cells in vitro and the therapeutic efficacy of L1-**11A** against i.p. SKOV3i.p. **tumor growth** in nude mice. In vitro, both anti-L1 **antibodies** efficiently inhibited the **proliferation** of SKOV3i.p. cells as well as other L1-expressing **tumor** cell lines (renal **carcinoma**, neuroblastoma, and colon **carcinoma**). On two cell lines, hyper-crosslinking of L1-**11A** with a secondary **antibody** was necessary for significant inhibition of **proliferation**, indicating that crosslinking of L1 is required for the antiproliferative effect. L1-neg. prostate **carcinoma** cells were not influenced by **antibody** treatment. Biweekly treatment of ovarian **carcinoma**-bearing mice with L1-**11A** led to a dose-dependent and significant reduction of **tumor** burden (up to -63.5%) and ascites formation (up to -75%). This effect was associated with reduced **proliferation** within the **tumors**. L1-directed **antibody**-based inhibition of peritoneal **growth** and dissemination of human ovarian **carcinoma** cells represents important proof-of-principle for the development of a new therapy against one of the leading gynecol. malignant diseases.
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2005:324328 CAPLUS
DN 142:390444
TI Gene expression profiles for classification of estrogen receptor status, diagnosis, and prognosis of breast **cancer**
IN Yu, Kun; Tan, Patrick
PA NCC Technology Ventures Pte. Limited, Singapore; Forrest, Graham R.
SO PCT Int. Appl., 153 pp.
CODEN: PIXXD2
DT Patent

LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005033336	A2	20050414	WO 2004-GB4190	20041001
	WO 2005033336	A3	20050929		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI GB 2003-23226 A 20031003

AB Classification of breast **tumors** into estrogen receptor pos. and neg. (ER+ and ER-) subtypes is an important distinction in the treatment of breast **cancer**. ER typing is frequently performed using expression profiles of genes whose expression is known to be affected by ER activity. Some **tumors** cannot confidently be assigned to a particular ER type based on such expression data. The present inventors have found that such 'low confidence' **tumors** constitute a distinct biol. subtype of breast **tumors** associated with significantly worse overall survival than high confidence **tumors** . Gene sets capable of distinguishing low confidence from high confidence **tumors** are provided, along with methods and apparatus for performing appropriate classification. of breast **tumors**. Although initially derived through purely computational means, the distinction between 'high' and 'low' confidence **tumors** is clin. meaningful, as 'low-confidence' **tumors** exhibit a significantly worse overall survival and shorter time to distant metastasis than their 'high-confidence' counterparts. Such a distinction is not currently discernible by conventional immunohistochem. strategies used to detect ER. A significant proportion of the "perturbed' genes are not known to be estrogen responsive and do not contain potential estrogen-response elements in their promoters. Further, high expression levels of the ERBB2 receptor are significantly correlated with breast **tumors** exhibiting a 'low confidence' prediction.

L16 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2005:141260 CAPLUS
DN 142:216625
TI Gene expression profiles associated with responses to neuropathic pain and their diagnostic and therapeutic uses
IN Tong, Jiefei; Jin, Gang; Ji, Rui-Ru; Xu, Yixun; Chiang, Lillian W.; Lavery, Daniel J.
PA Euro-Celtique, S. A., Luxembourg
SO PCT Int. Appl., 173 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005014849	A2	20050217	WO 2004-US23166	20040706

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	US 2005222027	A1	20051006	US 2004-989891	20041112
PRAI	US 2003-485101P	P	20030703		
	WO 2004-US23166	A2	20040706		

AB The present invention is based on gene expression profiles obtained from a spinal nerve ligation (SNL) model of neuropathic pain comprising tightly ligating the L5 and L6 spinal nerves in the rat. The rat SNL model is shown to be a valid model of neuropathic pain. Two hundred forty-nine differentially regulated genes are identified using the Affymetrix Rat U34 A, B and C arrays containing probesets representing .apprx.26,000 genes, including more than 1200 cDNAs (corresponding to mRNA) that are of known relevance to the field of neurobiol. The nucleic acids representing genes are subdivided into transcript classes representing functionally related proteins using gene expression herarchical clustering algorithms. By using these algorithms, the functional relevance of regulated genes was determined based on their gene expression data not only from the apparent up- or down-regulation between two conditions or a few conditions, but also from their entire expression pattern across 16 conditions in the animal pain model and expression distribution across 12 normal tissues, or 28 total conditions. GenBank identifiers and actual sequences corresponding to the human, mouse, and rat RefSeq top hits are identified for the 249 differentially regulated genes. The genes and their protein products can be used in screening methods to identify agonists and antagonists for the gene or gene product as potential therapeutic candidates.

L16 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2005:34707 CAPLUS
DN 142:128580
TI Prognosis determination in Ewing sarcoma patients by genetic profiling
IN Avigad, Smadar; Yaniv, Isaac; Zaizov, Rina; Ohali, Anat
PA Mor Research Applications Ltd., Israel
SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005002414	A2	20050113	WO 2004-IL578	20040630
	WO 2005002414	A3	20050310		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	EP 1641940	A2	20060405	EP 2004-744918	20040630

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK

PRAI	US 2003-483626P	P	20030701		
	WO 2004-IL578	W	20040630		

AB The present invention provides a method for assessing the prognosis of Ewing's sarcoma (ES) patients comprising determining the expression pattern of a defined set of genes in tumor material obtained from said

patients, and assigning said expression pattern to either a good prognosis or poor prognosis group. It is possible to distinguish between ES patients having a good prognosis and those having a poor prognosis by comparing gene expression patterns in nucleic acid material isolated from the **tumors**. Furthermore, this prognosis determination may be performed very early on, during initial diagnosis. Human Genome U95Av2 GeneChip microarrays (Affymetrix) were used to identify 818 genes differentially expressed in either the high-risk or the low-risk groups of 14 **tumor** samples, 7 **tumors** from patients who had progressed between 5 mo up to 5 years from diagnosis (defined as high-risk) and 7 **tumors** from patients who were disease-free for a low period of follow-up (defined as low-risk).

L16 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:409169 CAPLUS
DN 138:380506
TI Genes that are differentially expressed during erythropoiesis and their diagnostic and therapeutic uses
IN Brissette, William H.; Neote, Kuldeep S.; Zagouras, Panayiotis; Zenke, Martin; Lemke, Britt; Hacker, Christine
PA Pfizer Products Inc., USA; Max-Delbrueck-Centrum Fuer Molekulare Medizin
SO PCT Int. Appl., 285 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003038130	A2	20030508	WO 2002-XA34888	20021031
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	WO 2003038130	A2	20030508	WO 2002-US34888	20021031
	WO 2003038130	A3	20040212		
	WO 2003038130	C1	20040422		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRAI	US 2001-335048P	P	20011031		
	US 2001-335183P	P	20011102		
	WO 2002-US34888	A	20021031		

AB The present invention provides mol. targets that regulate erythropoiesis. Groups of genes or their encoded gene products comprise panels of the invention and may be used in therapeutic intervention, therapeutic agent screening, and in diagnostic methods for diseases and/or disorders of erythropoiesis. The panels were discovered using gene expression profiling of erythroid progenitors with Affymetrix HU6800 and HG-U95Av2 chips. Cells from an in vitro **growth** and differentiation system of SCF-Epo dependent human erythroid progenitors, E-cadherin+/CD36+

progenitors, cord blood, or CD34+ peripheral blood stem cells were analyzed. The HU6800 chip contains probes from 13,000 genes with a potential role in cell **growth**, **proliferation**, and differentiation and the HG-U95Av2 chip contains 12,000 full-length, functionally-characterized genes. [This abstract record is one of two records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L16 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:828415 CAPLUS
DN 137:89412
TI Detection of variations in the DNA methylation profile of genes in the determining the risk of disease
IN Berlin, Kurt; Piepenbrock, Christian; Olek, Alexander
PA Epigenomics A.-G., Germany
SO PCT Int. Appl., 636 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 69

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001077373	A2	20011018	WO 2001-XA1486	20010406
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, CF, CG, CI, CM, GA, GW, ML, MR, NE, SN, TD, TG				
	DE 10019058	A1	20011220	DE 2000-10019058	20000406
	WO 2001077373	A2	20011018	WO 2001-DE1486	20010406
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 2001077487	A5	20011023	AU 2001-77487	20010406
	EP 1360319	A2	20031112	EP 2001-955278	20010406
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	US 2004067491	A1	20040408	US 2003-240454	20030311
	AU 2003204553	A1	20040108	AU 2003-204553	20030605
	JP 2004008217	A2	20040115	JP 2003-160375	20030605
	US 2004023279	A1	20040205	US 2003-455212	20030605
PRAI	DE 2000-10019058	A	20000406		
	WO 2001-DE1486	W	20010406		
	DE 2000-10019173	A	20000407		
	DE 2000-10032529	A	20000630		
	DE 2000-10043826	A	20000901		
	WO 2001-EP4016	W	20010406		
	EP 2002-90203	A	20020605		

AB The invention relates to an oligonucleotide kit as probe for the detection of relevant variations in the DNA methylation of a target group of genes. The invention further relates to the use of the same for determining the gene variant with regard to DNA methylation, a medical device, using an oligonucleotide kit, a method for determining the methylation state of an individual and a method for the establishment of a model for establishing the probability of onset of a disease state in an individual. Such

diseases may be: undesired pharmaceutical side-effects; cancerous diseases; CNS dysfunctions, injuries or diseases; aggressive symptoms or relational disturbances; clin., psychol. and social consequences of brain injury; psychotic disorders and personality disorders; dementia and/or associated syndromes; cardiovascular disease, dysfunction and damage; dysfunction, damage or disease of the gastrointestinal tract; dysfunction, damage or disease of the respiratory system; injury, inflammation, infection, immunity and/or anastasis; dysfunction, damage or disease of the body as an abnormal development process; dysfunction, damage or disease of the skin, muscle, connective tissue or bones; endocrine and metabolic dysfunction, damage or disease; headaches or sexual dysfunction.
This abstract record is one of several records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.

L16 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2000:881321 CAPLUS
DN 134:38630
TI Streptavidin expressed gene fusions forming tetrameric complexes with therapeutic implications for adenocarcinomas and hematol. malignancies
IN Goshorn, Stephen Charles; Graves, Scott Stoll; Schultz, Joanne Elaine; Lin, Yukang; Sanderson, James Allen; Reno, John M.
PA Neorx Corp., USA
SO PCT Int. Appl., 99 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 5

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000075333	A1	20001214	WO 2000-US15595	20000605
	WO 2000075333	C2	20020620		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2376192	AA	20001214	CA 2000-2376192	20000605
	AU 2000055975	A5	20001228	AU 2000-55975	20000605
	EP 1190061	A1	20020327	EP 2000-941246	20000605
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	JP 2003501096	T2	20030114	JP 2001-502595	20000605
PRAI	US 1999-137900P	P	19990607		
	US 1999-168976P	P	19991203		
	WO 2000-US15595	W	20000605		

AB The present invention provides vectors for expressing genomic streptavidin fusion cassettes which include inducible promoters and various linkers and signal sequences. In the various embodiments, fusion proteins produced from these vectors are provided. In particular embodiments, fusion proteins comprising a single chain **antibody** (huNR-LU-10) and genomic streptavidin are provided as are vectors encoding the same. Also provided, are methods of using the fusion proteins of the present invention, and in particular, the use of scFvSA fusion proteins involving B9E9 as diagnostic markers or as a cell specific targeting agents. In addition tetravalent **antibodies** that contact a fusion protin forming a tetrametric complex which may comprise a **tumor** cell surface-associated protein and a streptavidin portion capable of binding biotin and a biotinylated radionuclide containing compound A immunoreactivity assay is described in addition to monitoring of blood clearance and

tumor uptake of fusion proteins. Some adenocarcinomas and hematol. malignancies such as non-Hodgkin's lymphoma may be treated with these fusio-protein expressing vectors. This system offers the expression of a genomic streptavidin gene fusion as a soluble protein into the periplasmic space of Escherichia coli where it undergoes spontaneous folding. This expression offers efficient protein folding where one does not need to purify and refold the protein expressed.

RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2
AN 1995:942163 CAPLUS
DN 124:27733
TI Regulation of interleukin 6 in multiple myeloma and bone marrow stromal cells
AU Chauhan, Dharminder; Uchiyama, Hiroshi; Urashima, Mitsuyoshi; Yamamoto, Ken-ichi; Anderson, Kenneth C.
CS Dana-Farber Cancer Institute, Harvard Medical School, Boston, MA, 02115, USA
SO Stem Cells (Dayton) (1995), 13(Suppl. 2), 35-9
CODEN: STCEEJ; ISSN: 1066-5099
PB AlphaMed Press
DT Journal
LA English
AB We and others have shown that some freshly isolated multiple myeloma (MM) cells and derived cell lines express interleukin 6 (IL-6) receptors and proliferate in vitro in response to IL-6; a subset of MM cells also expresses IL-6 mRNA, is intracytoplasmic IL-6 pos. and secretes IL-6. We have shown that MM cells express the cell surface adhesion mols. CD29/CDw49d(VLA-4), CD18/CD11a(LFA-1) and CD44, and may localize to marrow via specific adherence to both extracellular matrix proteins and to bone marrow stromal cells (BMSCs). MM **cell adhesion** triggers IL-6 secretion by normal and MM BMSCs and related IL-6-mediated **tumor** cell **growth**. Our attempts to block MM **cell adhesion** to BMSC-induced IL-6 secretion by using **antibodies** to CD29/CDw49d, CD18/**11a**, and/or CD44 demonstrated minimal effects, suggesting that another ligand-receptor interaction triggers IL-6 secretion when MM cells and BMSCs are juxtaposed. Both MM cells and BMSCs express CD40. Triggering of MM cells and BMSCs via CD40 upregulates IL-6 secretion in both MM cells and MM-derived cell lines, as well as BMSCs and BMSC lines, suggesting the possibility of both autocrine and paracrine MM cell **growth** triggered via CD40. Finally, expts. using the LP 101 BMSC line transiently transfected with IL-6 promoter fragments linked to chloramphenicol acetyltransferase reporter gene demonstrate that adhesion of MM cells induces IL-6 gene transcription in BMSCs, which is conferred via the NF-kB binding motif. Further characterization of mechanism of IL-6 regulation in MM cells and BMSCs may provide new therapeutic strategies based upon interruption of IL-6-mediated autocrine and paracrine **tumor** cell **growth**.

=> d l18 bib abs 1-20

L18 ANSWER 1 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2006:29606 CAPLUS
DN 144:121754
TI Gene expression profile for predicting activity of compounds that interact with and/or modulate protein tyrosine kinases and/or protein tyrosine pathways in lung **cancer cells**
IN Huang, Fei; Reeves, Karen A.; Han, Xia; Fairchild, Craig R.; Shaw, Peter
PA Bristol-Myers Squibb Company, USA
SO PCT Int. Appl., 130 pp.
CODEN: PIXXD2

DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006005035	A2	20060112	WO 2005-US23687	20050629

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

US 2006019284	A1	20060126	US 2005-169041	20050628
PRAI US 2004-584405P	P	20040630		

AB The present invention describes polynucleotides that have been discovered to correlate to the relative intrinsic sensitivity or resistance of **cells**, e.g., lung **cell** lines, to treatment with compds. that interact with and modulate, e.g., inhibit, protein tyrosine kinases, such as, for example, members of the Src family of tyrosine kinases, e.g., Src, Fgr, Fyn, Yes, Blk, Hck, Lck and Lyn, as well as other protein tyrosine kinases, including, Bcr-abl, Jak, PDGFR, c-kit and Ephr. These polynucleotides have been shown, through a weighted voting cross validation program, to have utility in predicting the resistance and sensitivity of lung **cell** lines to the compds. The expression level of some polynucleotides is regulated by treatment with a particular protein tyrosine kinase inhibitor compound, thus indicating that these polynucleotides are involved in the protein tyrosine kinase signal transduction pathway, e.g., Src tyrosine kinase. The Affymetrix human HG-U133 GeneChip set of over 44,792 probe sets was used to identify 129 polynucleotides that are highly correlated with a resistance/sensitivity phenotype classification of 23 lung **cell** lines subjected to treatment with the protein tyrosine kinase inhibitor compound BMS-A. Of the 129 predictor polynucleotides, 81 polynucleotides highly expressed in the **cell** lines were classified as sensitive to BMS-A, while 48 polynucleotides highly expressed in the **cell** lines were classified as resistant to BMS-A. Such polynucleotides, whose expression levels correlate highly with drug sensitivity or resistance and which are modulated by treatment with the compds., comprise polynucleotide predictor or marker sets useful in methods of predicting drug response, and as prognostic or diagnostic indicators in disease management, particularly in those disease areas, e.g., lung **cancer**, in which signaling through the protein tyrosine kinase pathway, such as the Src tyrosine kinase pathway, is involved with the disease process.

L18 ANSWER 2 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2006:338002 CAPLUS
DN 144:383459
TI Screening parkinson's disease therapeutics based on genes differentially expressed in A9 dopaminergic neurons
IN Isacson, Ole
PA USA
SO U.S. Pat. Appl. Publ., 35 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI US 2006078890 A1 20060413 US 2004-962080 20041008
WO 2006042137 A2 20060420 WO 2005-US36208 20051007
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
PRAI US 2004-962080 A 20041008
AB The present invention features methods of identifying compds. useful for the treatment and prevention of Parkinson's disease (PD). The invention is based on our discovery of numerous genes that are differentially expressed in A9 dopaminergic neurons, which undergo a disproportionately high level of **cell** death in PD, compared to A10 dopaminergic neurons, which are relatively spared. Compds. that reduce or prevent neurodegeneration caused by PD can be identified using screening methods that employ the genes and/or polypeptides that are differentially expressed in neurodegeneration-sensitive (A9) and neurodegeneration-resistant (A10) **cells**. Screening methods that make use of a plurality of such genes and polypeptides allow for the identification of agents associated with an improved ability to specifically and effectively treat and prevent neurodegeneration. Microarray anal. was performed to investigate the mol. differences between dopaminergic neurons located in the A9 and A10 midbrain regions. The differences that distinguished these two neuronal populations illustrated that only a small number of genes were differentially expressed. Forty-six genes had greater than 2.0-fold elevation of mRNA levels in A9 compared to A10 DA neurons, and 199 genes, greater than 1.5-fold [false discovery rate (FDR)<5 %]. Sixty-one genes had greater than 2.0-fold elevation of mRNA level in A10 compared to A9 DA neurons and 163 genes, greater than 1.5 fold (FDR<5 %) (Tables 4 and 5).

L18 ANSWER 3 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1
AN 2006:55798 CAPLUS
DN 144:127146
TI Efficient Inhibition of Intra-Peritoneal Tumor Growth and Dissemination of Human Ovarian Carcinoma **Cells** in Nude Mice by Anti-L1-**Cell Adhesion** Molecule Monoclonal **Antibody** Treatment
AU Arlt, Matthias J. E.; Novak-Hofer, Ilse; Gast, Daniela; Gschwend, Verena; Moldenhauer, Gerhard; Gruenberg, Juergen; Honer, Michael; Schubiger, P. August; Altevogt, Peter; Krueger, Achim
CS Klinikum rechts der Isar, Technischen Universitaet Muenchen, Munich, Germany
SO Cancer Research (2006), 66(2), 936-943
CODEN: CNREA8; ISSN: 0008-5472
PB American Association for Cancer Research
DT Journal
LA English
AB The L1 **cell adhesion** mol. is implicated in the control of proliferation, migration, and invasion of several tumor **cell** types in vitro. Recently, L1 overexpression was found to correlate with tumor progression of ovarian carcinoma, one of the most common causes of cancer-related deaths in gynecol. malignant diseases. To evaluate L1 as a potential target for ovarian cancer therapy, the authors investigated the effects of anti-L1 monoclonal **antibodies** (chCE7 and L1-**11A**) on proliferation and migration of L1-pos. human SKOV3i.p. ovarian carcinoma **cells** in vitro and the therapeutic efficacy of L1-**11A** against i.p. SKOV3i.p. tumor growth in nude mice. In

vitro, both anti-L1 **antibodies** efficiently inhibited the proliferation of SKOV3i.p. **cells** as well as other L1-expressing tumor **cell** lines (renal carcinoma, neuroblastoma, and colon carcinoma). On two **cell** lines, hyper-crosslinking of L1-**11A** with a secondary **antibody** was necessary for significant inhibition of proliferation, indicating that crosslinking of L1 is required for the antiproliferative effect. L1-neg. prostate carcinoma **cells** were not influenced by **antibody** treatment. Biweekly treatment of ovarian carcinoma-bearing mice with L1-**11A** led to a dose-dependent and significant reduction of tumor burden (up to -63.5%) and ascites formation (up to -75%). This effect was associated with reduced proliferation within the tumors. L1-directed **antibody**-based inhibition of peritoneal growth and dissemination of human ovarian carcinoma **cells** represents important proof-of-principle for the development of a new therapy against one of the leading gynecol. malignant diseases.

RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 4 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2005:324328 CAPLUS
DN 142:390444
TI Gene expression profiles for classification of estrogen receptor status, diagnosis, and prognosis of breast **cancer**
IN Yu, Kun; Tan, Patrick
PA NCC Technology Ventures Pte. Limited, Singapore; Forrest, Graham R.
SO PCT Int. Appl., 153 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005033336	A2	20050414	WO 2004-GB4190	20041001
	WO 2005033336	A3	20050929		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI GB 2003-23226 A 20031003

AB Classification of breast **tumors** into estrogen receptor pos. and neg. (ER+ and ER-) subtypes is an important distinction in the treatment of breast **cancer**. ER typing is frequently performed using expression profiles of genes whose expression is known to be affected by ER activity. Some **tumors** cannot confidently be assigned to a particular ER type based on such expression data. The present inventors have found that such 'low confidence' **tumors** constitute a distinct biol. subtype of breast **tumors** associated with significantly worse overall survival than high confidence **tumors**. Gene sets capable of distinguishing low confidence from high confidence **tumors** are provided, along with methods and apparatus for performing appropriate classification. of breast **tumors**. Although initially derived through purely computational means, the distinction between 'high' and 'low' confidence **tumors** is clin. meaningful, as 'low-confidence' **tumors** exhibit a significantly worse overall survival and shorter time to distant metastasis than their 'high-confidence' counterparts. Such a distinction is not currently

discernible by conventional immunohistochem. strategies used to detect ER. A significant proportion of the "perturbed' genes are not known to be estrogen responsive and do not contain potential estrogen-response elements in their promoters. Further, high expression levels of the ERBB2 receptor are significantly correlated with breast **tumors** exhibiting a 'low confidence' prediction.

L18 ANSWER 5 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2005:141260 CAPLUS
DN 142:216625
TI Gene expression profiles associated with responses to neuropathic pain and their diagnostic and therapeutic uses
IN Tong, Jiefei; Jin, Gang; Ji, Rui-Ru; Xu, Yixun; Chiang, Lillian W.; Lavery, Daniel J.
PA Euro-Celtique, S. A., Luxembourg
SO PCT Int. Appl., 173 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005014849	A2	20050217	WO 2004-US23166	20040706
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	US 2005222027	A1	20051006	US 2004-989891	20041112
PRAI	US 2003-485101P	P	20030703		
	WO 2004-US23166	A2	20040706		

AB The present invention is based on gene expression profiles obtained from a spinal nerve ligation (SNL) model of neuropathic pain comprising tightly ligating the L5 and L6 spinal nerves in the rat. The rat SNL model is shown to be a valid model of neuropathic pain. Two hundred forty-nine differentially regulated genes are identified using the Affymetrix Rat U34 A, B and C arrays containing probesets representing .apprx.26,000 genes, including more than 1200 cDNAs (corresponding to mRNA) that are of known relevance to the field of neurobiol. The nucleic acids representing genes are subdivided into transcript classes representing functionally related proteins using gene expression herarchical clustering algorithms. By using these algorithms, the functional relevance of regulated genes was determined based on their gene expression data not only from the apparent up- or down-regulation between two conditions or a few conditions, but also from their entire expression pattern across 16 conditions in the animal pain model and expression distribution across 12 normal tissues, or 28 total conditions. GenBank identifiers and actual sequences corresponding to the human, mouse, and rat RefSeq top hits are identified for the 249 differentially regulated genes. The genes and their protein products can be used in screening methods to identify agonists and antagonists for the gene or gene product as potential therapeutic candidates.

L18 ANSWER 6 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2005:34707 CAPLUS
DN 142:128580
TI Prognosis determination in Ewing sarcoma patients by genetic profiling
IN Avigad, Smadar; Yaniv, Isaac; Zaizov, Rina; Ohali, Anat
PA Mor Research Applications Ltd., Israel

SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005002414	A2	20050113	WO 2004-IL578	20040630
WO 2005002414	A3	20050310		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1641940	A2	20060405	EP 2004-744918	20040630

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK

PRAI US 2003-483626P	P	20030701
WO 2004-IL578	W	20040630

AB The present invention provides a method for assessing the prognosis of Ewing's sarcoma (ES) patients comprising determining the expression pattern of a defined set of genes in **tumor** material obtained from said patients, and assigning said expression pattern to either a good prognosis or poor prognosis group. It is possible to distinguish between ES patients having a good prognosis and those having a poor prognosis by comparing gene expression patterns in nucleic acid material isolated from the **tumors**. Furthermore, this prognosis determination may be performed very early on, during initial diagnosis. Human Genome U95Av2 GeneChip microarrays (Affymetrix) were used to identify 818 genes differentially expressed in either the high-risk or the low-risk groups of 14 **tumor** samples, 7 **tumors** from patients who had progressed between 5 mo up to 5 years from diagnosis (defined as high-risk) and 7 **tumors** from patients who were disease-free for a low period of follow-up (defined as low-risk).

L18 ANSWER 7 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2004:126643 CAPLUS
DN 141:22076
TI Lymphocyte Homing in Xenotransplanted Human Thyroid Tissue Can Be Inhibited by LFA-1 and ICAM-1 **Antibodies**
AU Jungheim, K.; Caspar, G.; Usadel, K. H.; Schumm-Draeger, P. M.
CS Center of Internal Medicine, Department of Medicine I, J.W. Goethe-University, Frankfurt, Germany
SO Thyroid (2004), 14(1), 3-11
CODEN: THYRER; ISSN: 1050-7256
PB Mary Ann Liebert, Inc.
DT Journal
LA English
AB Objectives: Homing of lymphocytes is an important factor with respect to the initiation of the autoimmune process in Graves' disease (GD). As previously shown, human lymphocytes, particularly of intrathyroidal origin, derived from patients with GD, are able to migrate into normal xenotransplanted thyroid tissue and induce functional and histol. changes. The aim of this study was to investigate the effect of LFA-1 and ICAM-1 **antibodies** on the homing of lymphocytes of different origin into xenografted human thyroid tissue. Methods: Eighty-five nude mice bearing 8-wk-old xenografts of normal human thyroid tissue were treated twice with

anti-CD 54 (anti-ICAM-1), anti-CD **11a** (anti-LFA-1), a combination of both, or, serving as controls, iso-**antibodies** without specific binding capacity or saline. Thereafter, intrathyroidal (ITL) or peripheral blood lymphocytes (PBL) obtained from 4 patients with GD or saline were injected into the animals (i.v., 0.2 mL, 106 **cells**). After 48 h the mice were sacrificed and transplants as well as mice thyroids were examined by immunohistochem. staining with Ki67, CD3, HLA-II (DAKO, Hamburg), IgG, CD44, ICAM-1, and VCAM-1 (Immunotech, Hamburg). Results: Pretreatment with anti-ICAM-1 and anti-LFA-1 decreased lymphocyte homing (CD3-staining), and expression of HLA-II, IgG, CD44, and VCAM-1 in the transplants. Conclusion: Our data show that [ICAM-1/LFA-1 stimulated (induced)] lymphocyte homing and subsequently thyrocyte proliferation are inhibited by ICAM-1 and LFA-1 **antibodies** in xenotransplanted thyroid tissue. This suggests that ICAM1 and LFA-1 play an important role in the early steps of autoimmune thyroid disease. The inhibition/suppression of ICAM-1 and LFA-1 interaction by resp. **antibodies**, as demonstrated in the present study, may provide a new concept for prophylaxis and therapy.

RE.CNT 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 8 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:409169 CAPLUS
DN 138:380506
TI Genes that are differentially expressed during erythropoiesis and their diagnostic and therapeutic uses
IN Brissette, William H.; Neote, Kuldeep S.; Zagouras, Panayiotis; Zenke, Martin; Lemke, Britt; Hacker, Christine
PA Pfizer Products Inc., USA; Max-Delbrueck-Centrum Fuer Molekulare Medizin
SO PCT Int. Appl., 285 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003038130	A2	20030508	WO 2002-XA34888	20021031
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	WO 2003038130	A2	20030508	WO 2002-US34888	20021031
	WO 2003038130	A3	20040212		
	WO 2003038130	C1	20040422		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRAI	US 2001-335048P	P	20011031		
	US 2001-335183P	P	20011102		
	WO 2002-US34888	A	20021031		

AB The present invention provides mol. targets that regulate erythropoiesis. Groups of genes or their encoded gene products comprise panels of the invention and may be used in therapeutic intervention, therapeutic agent screening, and in diagnostic methods for diseases and/or disorders of erythropoiesis. The panels were discovered using gene expression profiling of erythroid progenitors with Affymetrix HU6800 and HG-U95Av2 chips. **Cells** from an in vitro **growth** and differentiation system of SCF-Epo dependent human erythroid progenitors, E-cadherin+/CD36+ progenitors, cord blood, or CD34+ peripheral blood stem **cells** were analyzed. The HU6800 chip contains probes from 13,000 genes with a potential role in **cell growth**, **proliferation**, and differentiation and the HG-U95Av2 chip contains 12,000 full-length, functionally-characterized genes. [This abstract record is one of two records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L18 ANSWER 9 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:828415 CAPLUS
DN 137:89412
TI Detection of variations in the DNA methylation profile of genes in the determining the risk of disease
IN Berlin, Kurt; Piepenbrock, Christian; Olek, Alexander
PA Epigenomics A.-G., Germany
SO PCT Int. Appl., 636 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 69

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001077373	A2	20011018	WO 2001-XA1486	20010406
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, CF, CG, CI, CM, GA, GW, ML, MR, NE, SN, TD, TG				
	DE 10019058	A1	20011220	DE 2000-10019058	20000406
	WO 2001077373	A2	20011018	WO 2001-DE1486	20010406
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 2001077487	A5	20011023	AU 2001-77487	20010406
	EP 1360319	A2	20031112	EP 2001-955278	20010406
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	US 2004067491	A1	20040408	US 2003-240454	20030311
	AU 2003204553	A1	20040108	AU 2003-204553	20030605
	JP 2004008217	A2	20040115	JP 2003-160375	20030605
	US 2004023279	A1	20040205	US 2003-455212	20030605
PRAI	DE 2000-10019058	A	20000406		
	WO 2001-DE1486	W	20010406		
	DE 2000-10019173	A	20000407		
	DE 2000-10032529	A	20000630		
	DE 2000-10043826	A	20000901		

WO 2001-EP4016 W 20010406
EP 2002-90203 A 20020605

AB The invention relates to an oligonucleotide kit as probe for the detection of relevant variations in the DNA methylation of a target group of genes. The invention further relates to the use of the same for determining the gene variant with regard to DNA methylation, a medical device, using an oligonucleotide kit, a method for determining the methylation state of an individual and a method for the establishment of a model for establishing the probability of onset of a disease state in an individual. Such diseases may be: undesired pharmaceutical side-effects; cancerous diseases; CNS dysfunctions, injuries or diseases; aggressive symptoms or relational disturbances; clin., psychol. and social consequences of brain injury; psychotic disorders and personality disorders; dementia and/or associated syndromes; cardiovascular disease, dysfunction and damage; dysfunction, damage or disease of the gastrointestinal tract; dysfunction, damage or disease of the respiratory system; injury, inflammation, infection, immunity and/or anastasis; dysfunction, damage or disease of the body as an abnormal development process; dysfunction, damage or disease of the skin, muscle, connective tissue or bones; endocrine and metabolic dysfunction, damage or disease; headaches or sexual dysfunction.
This abstract record is one of several records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.

L18 ANSWER 10 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:831767 CAPLUS
DN 137:88421
TI Genetic polymorphisms in genes associated with drug metabolism and their use in selecting drug therapies
IN Stanton, Vincent; Zillmann, Martin
PA USA
SO U.S. Pat. Appl. Publ., 210 pp., Cont.-in-part of U.S. Ser. No. 710,467.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 6

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2001034023	A1	20011025	US 2000-733000	20001207
	WO 2000050639	A2	20000831	WO 2000-US1392	20000120
	WO 2000050639	A3	20020510		
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 2001034023	A1	20011025	US 2000-733000	20001207
PRAI	US 1999-131334P	P	19990426		
	US 1999-139440P	P	19990615		
	WO 2000-US1392	W	20000120		
	US 2000-696482	A2	20001024		
	US 2000-710467	A2	20001108		
	US 2000-733000	A	20001207		
	US 1999-121047P	P	19990222		
	US 1999-357743	A	19990720		

AB Methods for identifying and utilizing variances in genes relating to efficacy and safety of medical therapy and other aspects of medical therapy are described, including methods for selecting an effective treatment. [This abstract record is one of several records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L18 ANSWER 11 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2000:881321 CAPLUS
DN 134:38630
TI Streptavidin expressed gene fusions forming tetrameric complexes with therapeutic implications for adenocarcinomas and hematol. malignancies
IN Goshorn, Stephen Charles; Graves, Scott Stoll; Schultz, Joanne Elaine; Lin, Yukang; Sanderson, James Allen; Reno, John M.
PA Neorx Corp., USA
SO PCT Int. Appl., 99 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 5

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000075333	A1	20001214	WO 2000-US15595	20000605
	WO 2000075333	C2	20020620		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2376192	AA	20001214	CA 2000-2376192	20000605
	AU 2000055975	A5	20001228	AU 2000-55975	20000605
	EP 1190061	A1	20020327	EP 2000-941246	20000605
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	JP 2003501096	T2	20030114	JP 2001-502595	20000605
PRAI	US 1999-137900P	P	19990607		
	US 1999-168976P	P	19991203		
	WO 2000-US15595	W	20000605		

AB The present invention provides vectors for expressing genomic streptavidin fusion cassettes which include inducible promoters and various linkers and signal sequences. In the various embodiments, fusion proteins produced from these vectors are provided. In particular embodiments, fusion proteins comprising a single chain **antibody** (huNR-LU-10) and genomic streptavidin are provided as are vectors encoding the same. Also provided, are methods of using the fusion proteins of the present invention, and in particular, the use of scFvSA fusion proteins involving B9E9 as diagnostic markers or as a **cell** specific targeting agents. In addition tetravalent **antibodies** that contact a fusion protin forming a tetrametric complex which may comprise a **tumor cell** surface-associated protein and a streptavidin portion capable of binding biotin and a biotinylated radionuclide containing compound A immunoreactivity assay is described in addition to monitoring of blood clearance and **tumor** uptake of fusion proteins. Some adenocarcinomas and hematol. malignancies such as non-Hodgkin's lymphoma may be treated with these fusio-protein expressing vectors. This system offers the expression of a genomic streptavidin gene fusion as a soluble protein into the periplasmic space of Escherichia coli where it undergoes spontaneous folding. This expression offers efficient protein folding where one does not need to purify and refold the protein expressed.
RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 12 OF 20 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN
AN 1998088530 ESBIOBASE
TI **Adhesion** molecules in iris biopsy specimens from patients with

uveitis
AU La Heij E.; Kuijpers R.W.A.; Baarsma S.G.; Kijlstra A.; Van der Weiden M.; Mooy C.M.
CS Dr. E. La Heij, Academisch Ziekenhuis Maastricht, Postbus 5800, 6202 AZ Maastricht, Netherlands.
SO British Journal of Ophthalmology, (1998), 82/4 (432-437), 28 reference(s) CODEN: BJOPAL ISSN: 0007-1161
DT Journal; Article
CY United Kingdom
LA English
SL English
AB Background/aims - Earlier studies on intraocular tissue have demonstrated that T lymphocytes play a major role in the pathogenesis of uveitis. **Adhesion** molecules are immunoregulatory molecules for the interaction between T lymphocytes and vascular endothelium and they play an important role in the recruitment of specific T lymphocytes from the circulation into inflamed tissue. In uveitis an increased expression of some of these **adhesion** molecules may be expected. Methods - The presence of **adhesion** molecules was investigated in iris biopsy specimens from 11 patients with uveitis and eight controls (patients with primary open angle glaucoma) immunohistochemically with a panel of monoclonal **antibodies**: LECAM (CD 62L), ICAM-1 (CD 54), LFA-1 (CD **11a**/18), VCAM-1 (CD 106), VLA-4 (CD 49d), and HECA-452, a marker for high endothelial venules. Results - Positive staining for ICAM-1, LFA-1 and VCAM-1 was found in the iris in a significantly higher number of uveitis patients than in controls. The remaining **adhesion** molecules were also found in a higher number of uveitis patients than in controls, but this difference did not reach statistical significance. Conclusion - An increased expression of **adhesion** molecules was found in the iris of patients with uveitis, indicating an immunoregulatory function for **adhesion** molecules in the pathogenesis of uveitis.

L18 ANSWER 13 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1998:362953 CAPLUS
DN 129:188130
TI Analysis of the immunological cross reactivities of 213 well characterized monoclonal **antibodies** with specificities against various leukocyte surface antigens of human and 11 animal species
AU Brodersen, R.; Bijlsma, F.; Gori, K.; Jensen, K. T.; Chen, W.; Dominguez, J.; Haverson, K.; Moore, P. F.; Saalmuller, A.; Sachs, D.; Slierendrecht, W. J.; Stokes, C.; Vainio, O.; Zuckermann, F.; Aasted, B.
CS Department of Veterinary Microbiology, Laboratory of Virology and Immunology, Royal Veterinary and Agricultural University, Copenhagen, 1870, Den.
SO Veterinary Immunology and Immunopathology (1998), 64(1), 1-14 CODEN: VIIMDS; ISSN: 0165-2427
PB Elsevier Science B.V.
DT Journal
LA English
AB 213 Monoclonal **antibodies** (mAbs) raised against leukocyte surface antigens from human and 11 animal species were analyzed for reactivities against leukocytes from human and 15 different animal species. We found 77 mAbs (36%) to cross-react. Altogether, 217 cross reactions were registered out of 3195 possible combinations (7%). Most of the cross reacting mAbs had integrin or MHC class II specificities. This study defined cross reactions on the following markers: CD1a, 1c, 2, 4, 5, 8, 9, **11a**, 11b, 14, 18, 20, 21, 23, 29, 31, 41, 43, 44, 45, 45R, 46, 49, 61, 62L, TCR γ/δ, BCR, Thy-1, MHC class I and MHC class II, Swine-WC7 and Cattle-WC1. In order to characterize the mol. weight (MW) of the corresponding cross reacting antigens, selected mAbs were used to immunoppt. the antigens. The MW's of the analyzed precipitated antigens were in good agreement with the MWs of the homologous antigens. The followed

strategy was found to be efficient and economical in defining new leukocyte antigen reactive mAbs.

RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 14 OF 20 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN
AN 1996185738 ESBIOBASE
TI Attenuation of rat lung isograft reperfusion injury with a combination of anti-ICAM-1 and anti-β.sub.2 integrin monoclonal **antibodies**
AU DeMeester S.R.; Molinari M.A.; Shiraishi T.; Okabayashi K.; Manchester J.K.; Wick M.R.; Cooper J.D.; Patterson G.A.
CS S.R. DeMeester, Division of Cardiothoracic Surgery, Department of Surgery, Washington Univ. School of Medicine, 1 Barnes Hospital Plaza, St. Louis, MO 63110, United States.
SO Transplantation, (1996), 62/10 (1477-1485)
CODEN: TRPLAU ISSN: 0041-1337
DT Journal; Article
CY United States
LA English
SL English
AB Four different combinations of monoclonal **antibodies** against rat ICAM- 1, CD-**11a**, and CD-18 were utilized to determine the relative importance of LFA-1, Mac-1, and ICAM-1 in a rat model of severe lung allograft reperfusion injury. Negative control animals were given phosphate buffered saline (the carrier solution for the **antibodies**), while positive control animals were rendered neutropenic by the administration of a polyclonal mouse IgG. **Antibodies** were given with the donor lung flush, prior to left lung graft reperfusion, or both. Isolated graft function was determined 24 hr after implantation by arterial blood gas (ABG), and after sacrifice the native and transplanted lungs underwent bronchoalveolar lavage for alveolar protein qnantitation, **cell** count and differential, and myeloperoxidase assay. Additionally, whole lung homogenates were assayed for myeloperoxidase activity. We found that the combination of anti-ICAM-1 (1 mg/kg) added to the donor lung flush, and anti-CD11a, anti-CD18, and anti-ICAM-1 (2 mg/kg i.v. of each) given to the recipient prior to reperfusion, resulted in significantly improved lung graft pAO.sub.2 by ABG, and decreased alveolar protein, **cell** count, and myeloperoxidase activity compared with control animals. Improvement was less than that seen in the neutropenic recipients, however. We conclude that LFA-1, Mac-1, and ICAM-1 are all important **adhesion** molecules in lung allograft reperfusion injury-yet even with **antibody** blockade of all three there are additional mechanisms allowing for neutrophil influx into the lungs.

L18 ANSWER 15 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1997:36166 CAPLUS
DN 126:58837
TI **Cell adhesion** molecule mediates endothelial **cell** injury caused by activated neutrophils
AU Murota, Sei-Itsu; Fujita, Hiroshi; Wakabayashi, Yoshiyuki; Morita, Ikuo
CS Department of Physiological Chemistry, Graduate School, Tokyo Medical and Dental University, Tokyo, Japan
SO Keio Journal of Medicine (1996), 45(3), 207-212
CODEN: KJMEA9; ISSN: 0022-9717
PB Keio University, School of Medicine
DT Journal
LA English
AB Addition of PMA (phorbol myristate acetate)-stimulated neutrophils to an endothelial **cell** monolayer caused a significant increase in the intracellular peroxide level of the endothelial **cells** after 15 min and endothelial **cell** injury after 5 h. Both the early and the late events were abolished in the presence of specific

antibodies against CD (cluster of differentiation) **11a**, CD11b, CD18 and ICAM (intercellular **adhesion** mol.) 1, but not CD11c. These **antibodies** affected neither the production of active oxygen species by the neutrophils nor the rate of **adhesion** of neutrophils to endothelial **cells**. Pretreatment of endothelial **cells** with allopurinol caused significant inhibition of both the early and the late events, suggesting that the binding of **adhesion** mols. may trigger the activation of XO (xanthine oxidase) of endothelial **cells**, and have the **cells** produce more hydrogen peroxide and ferrous ions, followed by producing more hydrogen peroxide. The hydrogen peroxide produced by endothelial **cells** themselves and by neutrophils may be converted to hydroxyl radicals by ferrous ions, which may cause lethal **cell** damage. Examination of XO activity in endothelial **cells** showed that the enzyme activity increased double within 15 min after the addition of PMA activated neutrophils. Monoclonal **antibodies** against CD11a and CD18 significantly inhibited the increased conversion of XD (xanthine dehydrogenase) to XO induced by PMA-activated neutrophils. Moreover, tyrosine kinase inhibitors also inhibited the increased conversion of XD to XO. These results indicate that the **adhesion** of activated neutrophils to endothelial **cells** via CD11a/CD18-ICAM-1 is involved in the conversion of XD to XO in endothelial **cells**, which results in endothelial **cell** injury.

L18 ANSWER 16 OF 20 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN
AN 1996:26318949 BIOTECHNO
TI A novel monoclonal **antibody** mNI-58A against the α-chain of leukocyte function-associated antigen-1 (LFA-1) blocks the homotypic **cell** aggregation and actively regulates morphological changes in the phorbol myristate acetate (PMA)-activated human monocyte-like **cell** line, U937
AU Ikewaki N.; Yamada A.; Sonoda A.; Inoko H.
CS Department of Microbiology, Kitasato University School Nursing, Kitasato 2-1-1,Sagamihara, Kanagawa 228, Japan.
SO Tissue Antigens, (1996), 48/3 (161-173)
CODEN: TSANA2 ISSN: 0001-2815
DT Journal; Article
CY Denmark
LA English
SL English
AB A monoclonal **antibody** (mAb), designated mNI-58A, was produced by immunizing mice with the lipopolysaccharide (LPS)-stimulated monocyte-like **cell** line, U937. The antigen defined by mNI-58A was widely expressed on various lymphoid **cells** and all **cell** lines examined except the erythroid **cell** line, K562. When the reactive patterns between mNI-58A and the mAbs to various human differentiation antigens (CD11a, CD11b, CD11c, CD14, CD16, CD18, CD23, CD28, CD29, CD31, CD43, CD44, CD45RA, CD50, CD54, CD58, CD80, CD102, CD106, HLA-class I and -class II antigen) were compared, that of mNI-58A was found to be similar to those of the leukocyte function-associated antigen-1 (LFA-1) mAbs. Using a competitive immunofluorescence binding assay it was found that the preincubation with one of the CD11a mAbs, 2F12 completely blocked the subsequent binding of mNI-58A. mNI-58A prevented the homotypic **cell** aggregation of the phorbol myristate acetate (PMA)-activated U937 **cells** (referred to as PMA-U937) and PMA-activated Epstein-Barr virus (EBV)-transformed B **cell** lines, B-85 and Mann, mNI-58A markedly induced the spread formation of the PMA-U937 **cells** following this blocking of the homotypic **cell** aggregation, whereas 2F12 did not under the same condition. The spread formation induced by mNI-58A was completely blocked by cytochalasin B (CyB), cytochalasin D (CyD), cycloheximide (CHX) or protein kinase C inhibitors, sphingosine and H-7. The U937 **cells** markedly adhered to the tumor necrosis factor-α (TNF-α)-stimulated human umbilical vein endothelial

cells (HUVECs) and also to the extracellular matrix protein, fibronectin, but mNI-58A did not enhance or block these **adhesion** processes. mNI-58A precipitated two glycoproteins with molecular weight 180 kDa and 95 kDa as determined by SDS-PAGE analysis, which were identical to the LFA-α (CD **11a**) and β (CD 18) chains of leukocyte integrin precipitated by the CD11a mAbs, respectively. Sequential immunoprecipitation studies using the CD11a mAb (2F12) also indicate that mNI-58A recognizes an epitope on the α-chain of the LFA-1 molecule. The ability of mNI-58A to block the PMA-U937 **cells** and to induce the spread formation of these **cells** suggests that mNI-58A is a novel mAb reacting with an epitope on the α-chain of LFA-1 different from those recognized with the existing CD **11a** mAbs.

L18 ANSWER 17 OF 20 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN
AN 1995034297 ESBIOBASE
TI Expression of **cell** interaction molecules by immature rat thymocytes during passage through the CD4.sup.+C8.sup.+ compartment: Developmental regulation and induction by t **cell** receptor engagement of CD2, CD5, CD28, CD11a, CD44 and CD53
AU Mitnacht R.; Tacke M.; Hunig T.
CS T. Hunig, Institut fur Virologie and Immunbiol, Universitat Wurzburg, Versbacher Strasse 7, D-97078 Wurzburg, Germany.
SO European Journal of Immunology, (1995), 25/2 (328-332)
CODEN: EJIMAF ISSN: 0014-2980
DT Journal; Article
CY Germany, Federal Republic of
LA English
SL English
AB Rat thymocytes of the T **cell** receptor(low) (TcR(low)) CD4.sup.+8.sup.+ subset: which is the target of repertoire selection are heterogeneous with respect to expression of the **cell** interaction (CI) molecules CD2, CD5, CD11a/CD18 (LFA-1), CB28 and CD44. We show that this heterogeneity is due to the developmental regulation of these CI molecules during passage through the CD4.sup.+8.sup.+ compartment, and to up-regulation by TcR engagement. Thus, cohorts of CD4.sup.+8.sup.+ **cells** differentiating synchronously in vitro from their direct precursors, the immature CD4.sup.-8.sup.+ **cells**, were homogeneous with regard to CI molecule expression. Upon entry into the CD4.sup.+8.sup.+ compartment, they expressed relatively high levels of CD2 and CD44, and moderate levels of CD5, CD28 and CD11a, CD2, CD28 and CD44 were slightly down-regulated during the following 2 days, whereas CD5 slightly increased and CD11a remained constant. TcR stimulation using immobilized monoclonal **antibodies** resulted in rapid and dramatic up-regulation of CD2, CD5 and CD28 and, to a lesser extent, of CD **11a** and CD44. Finally CD53, a triggering structure absent from unstimulated CD4.sup.+8.sup.+ thymocytes was also rapidly induced by TcR stimulation. Inclusion of interleukin (IL)-2, IL-4, or IL-7 in this in vitro differentiation system did not affect the levels of CI molecules studied. Since the high levels of CI molecules induced by TcR-stimulation correspond to those found in vivo on TcR(intermediate) thymocytes known to be undergoing repertoire selection, these results suggest that upregulation of CI molecules by TcR engagement provides a mechanism by which thymocytes that have entered the selection process gain preferential access to further interactions with stromal and lymphoid **cells** in the thymus.

L18 ANSWER 18 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2
AN 1995:942163 CAPLUS
DN 124:27733
TI Regulation of interleukin 6 in multiple myeloma and bone marrow stromal **cells**
AU Chauhan, Dharminder; Uchiyama, Hiroshi; Urashima, Mitsuyoshi; Yamamoto,

Ken-ichi; Anderson, Kenneth C.
CS Dana-Farber Cancer Institute, Harvard Medical School, Boston, MA, 02115, USA
SO Stem Cells (Dayton) (1995), 13(Suppl. 2), 35-9
CODEN: STCEEJ; ISSN: 1066-5099
PB AlphaMed Press
DT Journal
LA English
AB We and others have shown that some freshly isolated multiple myeloma (MM) **cells** and derived **cell** lines express interleukin 6 (IL-6) receptors and proliferate in vitro in response to IL-6; a subset of MM **cells** also expresses IL-6 mRNA, is intracytoplasmic IL-6 pos. and secretes IL-6. We have shown that MM **cells** express the **cell** surface **adhesion** mols. CD29/CDw49d(VLA-4), CD18/CD11a(LFA-1) and CD44, and may localize to marrow via specific adherence to both extracellular matrix proteins and to bone marrow stromal **cells** (BMSCs). MM **cell adhesion** triggers IL-6 secretion by normal and MM BMSCs and related IL-6-mediated tumor **cell** growth. Our attempts to block MM **cell adhesion** to BMSC-induced IL-6 secretion by using **antibodies** to CD29/CDw49d, CD18/**11a**, and/or CD44 demonstrated minimal effects, suggesting that another ligand-receptor interaction triggers IL-6 secretion when MM **cells** and BMSCs are juxtaposed. Both MM **cells** and BMSCs express CD40. Triggering of MM **cells** and BMSCs via CD40 upregulates IL-6 secretion in both MM **cells** and MM-derived **cell** lines, as well as BMSCs and BMSC lines, suggesting the possibility of both autocrine and paracrine MM **cell** growth triggered via CD40. Finally, expts. using the LP 101 BMSC line transiently transfected with IL-6 promoter fragments linked to chloramphenicol acetyltransferase reporter gene demonstrate that **adhesion** of MM **cells** induces IL-6 gene transcription in BMSCs, which is conferred via the NF-kB binding motif. Further characterization of mechanism of IL-6 regulation in MM **cells** and BMSCs may provide new therapeutic strategies based upon interruption of IL-6-mediated autocrine and paracrine tumor **cell** growth.

L18 ANSWER 19 OF 20 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 3
AN 1995:264803 CAPLUS
DN 122:53238
TI Immunohistological and functional analysis of **adhesion** molecule expression in the rheumatoid synovial lining layer. Implications for synovial lining **cell** destruction
AU Dinther-Janssen, Anna C. H. M. van; Kraal, George; Soesbergen, Rene M. van; Scheper, Rik J.; Meijer, Chris J. L. M.
CS Department Pathology and Department Histology, Free University and Slotervaart Hospital, Amsterdam, Neth.
SO Journal of Rheumatology (1994), 21(11), 1998-2004
CODEN: JRHUA9; ISSN: 0315-162X
DT Journal
LA English
AB It has previously been shown that the **adhesion** of lymphocytes to microvascular endothelium mediates lymphocyte extravasation within inflamed synovium. After passing the endothelial barrier, binding of lymphocytes to matrix proteins and synovial lining **cells** may further lead to synovial membrane hyperplasia and subsequent cartilage destruction. Thus, we have explored the mol. basis of T **cell** -synovial lining **cell** interaction in the synovial membrane of patients with rheumatoid arthritis (RA). Using an immunohistochem. staining technique and an in vitro frozen section assay we studied the expression and the role of several **adhesion** mols. in T lymphocyte-synovial lining **cell** interaction in the inflamed synovial membrane. In RA the macrophage-like (type A) synovial lining **cells** express high levels of intercellular **adhesion** mol.

1 [ICAM-1 (CD54)], whereas the fibroblast-like (type B) synovial lining **cells** predominantly express vascular **cell** **adhesion** mol. 1 (VCAM-1), in addition to moderate levels of ICAM-1. Both **cell** types express low levels of fibronectin. Unstimulated and anti-CD3 stimulated peripheral blood T **cells** bear the resp. ligands lymphocyte function associated antigen 1 [LFA-1 (CD18/**11a**)], and very late antigen 4 and 5 [VLA-4 (CD29/49d) and VLA-5 (CD29/49e)]. T lymphocytes predominantly bound to type B synovial lining **cells**. Inhibition studies with monoclonal **antibodies** revealed that this binding involves the VLA-4/VCAM-1 and VLA-5/fibronectin (FN), but not the VLA-4/CS1 pathway. LFA-1 is also involved in this interaction via its ligand ICAM-1. These results show that the mol. basis of T lymphocyte binding to rheumatoid synovial lining **cells** is different from that described for T lymphocyte binding to synovial membrane vascular endothelium which involves the VLA-4/VCAM-1 and VLA-4/CS-1 pathways, but not the LFA-1/ICAM-1 pathway.

L18 ANSWER 20 OF 20 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN
AN 1993:23048926 BIOTECHNO
TI **Adhesion** of precursor-B acute lymphoblastic leukaemia **cells** to bone marrow stromal proteins
AU Makrynikola V.; Bradstock K.F.
CS Haematology Department, Westmead Hospital,NSW 2145, Australia.
SO Leukemia, (1993), 7/1 (86-92)
CODEN: LEUKED ISSN: 0887-6924
DT Journal; Article
CY United Kingdom
LA English
SL English
AB **Adhesion** to bone marrow stroma is a key event in normal B lymphopoiesis, allowing exposure of B-**cell** progenitors to regulatory cytokines. In order to investigate whether similar processes are important in the proliferation of acute lymphoblastic leukaemia ALL) **cells** of precursor-B type, the expression of various **adhesion** molecules was examined. By flow cytometry analysis, CD-44 and the integrins VLA-4 and VLA-5 were the most prominent. CD-44 and VLA-4 were expressed on all 18 cases of precursor-B ALL analysed, while VLA-5 was found on 15 of 18 cases. The integrin CD-**11a** was detected on 8 of 11 cases, while its ligand, CD-54, was present in 6/12. Other **adhesion** proteins such as β3 integrin, CD-56, CD-15, and Leu8 were not expressed to any significant extent. In view of the known binding of VLA-4 and VLA-5 to extracellular fibronectin (FN), the **adhesion** of leukaemic **cells** to FN was evaluated in a colorimetric assay. The precursor-B ALL **cell** lines REH and KM-3, and 7/15 cases of precursor-B ALL, showed detectable binding to FN. Binding to the other extracellular matrix proteins collagen type 1 and vitronectin was not observed, although two ALL cases showed some binding to laminin. The functional activity of the VLA-4 and VLA-5 molecules was examined using an inhibitory peptide and monoclonal **antibodies**. These studies indicated that ALL **cells** adhere to soluble fibronectin predominantly through the VLA-5 molecule (blockable with the PHM-2 **antibody** and a peptide containing the RGD sequence) although binding mediated by VLA-4 was also apparent in some experiments (blockable by a 40 kDa fragment containing the heparin-binding domain of FN and inhibitory **antibodies**). These results indicate that precursor-B ALL **cells** may adhere to marrow stroma through interaction of VLA-4 and VLA-5 with FN, although other mechanisms of **adhesion** may be important.

=> d his

(FILE 'HOME' ENTERED AT 11:19:48 ON 19 MAY 2006)

```
     FILE 'CAPLUS, BIOENG, BIOTECHNO, BIOTECHDS, ESBIOBASE' ENTERED AT
     11:20:33 ON 19 MAY 2006
L1         23110 S  (((CELL (W) ADHESION) OR L1CAM) AND ANTIBODY)
L2          5521 S (L1 AND (PROLIFERATION OR GROWTH))
L3          2418 S (L2 AND (CANCER OR TUMOR OR TUMOUR OR NEOPLASIA OR CARCINOMA
L4         23103 S (L3 AND (L1 (W) ANTIGEN) OR (L1 (W) CELL (W) ADHESION))
L5             1 S (UJ127) AND L3
L6             5 S (UJ127) AND L4
L7             1 S L5 AND L6
L8             4 DUPLICATE REMOVE L6 (1 DUPLICATE REMOVED)
L9             1 S (5G3 AND L3)
L10            6 S (5G3 AND L4)
L11            4 DUPLICATE REMOVE L10 CAPLUS  (2 DUPLICATES REMOVED)
L12            4 DUPLICATE REMOVE L10 (2 DUPLICATES REMOVED)
L13           24 S ((L1 (W) 11A) AND ANTIBODY)
L14           13 S L13 AND L3
L15           24 S L13 AND L4
L16           10 DUPLICATE REMOVE L14 (3 DUPLICATES REMOVED)
L17           20 DUPLICATE REMOVE L15 CAPLUS  (4 DUPLICATES REMOVED)
L18           20 DUPLICATE REMOVE L15 (4 DUPLICATES REMOVED)

=> s (chCE7 and L3)
L19            5 (CHCE7 AND L3)

=> s (ceCE7 and L4)
L20            0 (CECE7 AND L4)

=> s (chCE7 and L4)
L21           11 (CHCE7 AND L4)

=> duplicate remove l19
DUPLICATE PREFERENCE IS 'CAPLUS, BIOENG, ESBIOBASE'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L19
L22            3 DUPLICATE REMOVE L19 (2 DUPLICATES REMOVED)

=> duplicate remove l21
DUPLICATE PREFERENCE IS 'CAPLUS, BIOENG, BIOTECHNO, ESBIOBASE'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L21
L23            6 DUPLICATE REMOVE L21 (5 DUPLICATES REMOVED)

=> d his

     (FILE 'HOME' ENTERED AT 11:19:48 ON 19 MAY 2006)

     FILE 'CAPLUS, BIOENG, BIOTECHNO, BIOTECHDS, ESBIOBASE' ENTERED AT
     11:20:33 ON 19 MAY 2006
L1         23110 S  (((CELL (W) ADHESION) OR L1CAM) AND ANTIBODY)
L2          5521 S (L1 AND (PROLIFERATION OR GROWTH))
L3          2418 S (L2 AND (CANCER OR TUMOR OR TUMOUR OR NEOPLASIA OR CARCINOMA
L4         23103 S (L3 AND (L1 (W) ANTIGEN) OR (L1 (W) CELL (W) ADHESION))
L5             1 S (UJ127) AND L3
L6             5 S (UJ127) AND L4
L7             1 S L5 AND L6
L8             4 DUPLICATE REMOVE L6 (1 DUPLICATE REMOVED)
L9             1 S (5G3 AND L3)
L10            6 S (5G3 AND L4)
L11            4 DUPLICATE REMOVE L10 CAPLUS  (2 DUPLICATES REMOVED)
L12            4 DUPLICATE REMOVE L10 (2 DUPLICATES REMOVED)
L13           24 S ((L1 (W) 11A) AND ANTIBODY)
L14           13 S L13 AND L3
L15           24 S L13 AND L4
L16           10 DUPLICATE REMOVE L14 (3 DUPLICATES REMOVED)
```

L17 20 DUPLICATE REMOVE L15 CAPLUS (4 DUPLICATES REMOVED)
L18 20 DUPLICATE REMOVE L15 (4 DUPLICATES REMOVED)
L19 5 S (CHCE7 AND L3)
L20 0 S (CECE7 AND L4)
L21 11 S (CHCE7 AND L4)
L22 3 DUPLICATE REMOVE L19 (2 DUPLICATES REMOVED)
L23 6 DUPLICATE REMOVE L21 (5 DUPLICATES REMOVED)

=> d l22 bib abs -13

L22 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1
AN 2006:55798 CAPLUS
DN 144:127146
TI Efficient Inhibition of Intra-Peritoneal **Tumor Growth** and Dissemination of Human Ovarian **Carcinoma** Cells in Nude Mice by Anti-L1-**Cell Adhesion** Molecule Monoclonal **Antibody** Treatment
AU Arlt, Matthias J. E.; Novak-Hofer, Ilse; Gast, Daniela; Gschwend, Verena; Moldenhauer, Gerhard; Gruenberg, Juergen; Honer, Michael; Schubiger, P. August; Altevogt, Peter; Krueger, Achim
CS Klinikum rechts der Isar, Technischen Universitaet Muenchen, Munich, Germany
SO Cancer Research (2006), 66(2), 936-943
CODEN: CNREA8; ISSN: 0008-5472
PB American Association for Cancer Research
DT Journal
LA English
AB The L1 **cell adhesion** mol. is implicated in the control of **proliferation**, migration, and invasion of several **tumor** cell types in vitro. Recently, L1 overexpression was found to correlate with **tumor** progression of ovarian **carcinoma** , one of the most common causes of **cancer**-related deaths in gynecol. malignant diseases. To evaluate L1 as a potential target for ovarian **cancer** therapy, the authors investigated the effects of anti-L1 monoclonal **antibodies** (**chCE7** and L1-11A) on **proliferation** and migration of L1-pos. human SKOV3i.p. ovarian **carcinoma** cells in vitro and the therapeutic efficacy of L1-11A against i.p. SKOV3i.p. **tumor growth** in nude mice. In vitro, both anti-L1 **antibodies** efficiently inhibited the **proliferation** of SKOV3i.p. cells as well as other L1-expressing **tumor** cell lines (renal **carcinoma**, neuroblastoma, and colon **carcinoma**). On two cell lines, hyper-crosslinking of L1-11A with a secondary **antibody** was necessary for significant inhibition of **proliferation**, indicating that crosslinking of L1 is required for the antiproliferative effect. L1-neg. prostate **carcinoma** cells were not influenced by **antibody** treatment. Biweekly treatment of ovarian **carcinoma**-bearing mice with L1-11A led to a dose-dependent and significant reduction of **tumor** burden (up to -63.5%) and ascites formation (up to -75%). This effect was associated with reduced **proliferation** within the **tumors**. L1-directed **antibody**-based inhibition of peritoneal **growth** and dissemination of human ovarian **carcinoma** cells represents important proof-of-principle for the development of a new therapy against one of the leading gynecol. malignant diseases.
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2004:292071 CAPLUS
DN 140:320040
TI 36Fusion proteins comprising CD1d complex, α2 microglobulin and **antibody** or fragment for targeting therapy of **tumor**, autoimmune disease, inflammation and infection
IN Robert, Bruno; Donda, Alena; Cesson, Valerie; Mach, Jean-Pierre; Zauderer,

Maurice
PA Vaccinex, Inc., USA
SO PCT Int. Appl., 152 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004029206	A2	20040408	WO 2003-US30238	20030926
	WO 2004029206	A3	20041007		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1413316	A1	20040428	EP 2002-405838	20020927
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
	CA 2502735	AA	20040408	CA 2003-2502735	20030926
	AU 2003275254	A1	20040419	AU 2003-275254	20030926
	EP 1551448	A2	20050713	EP 2003-759526	20030926
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
PRAI	EP 2002-405838	A	20020927		
	WO 2003-US30238	W	20030926		

AB The invention is directed to a compound comprising one or more CD1d complexes in association with an **antibody** specific for a cell surface marker. The CD1d complexes comprise a CD1d, a ss2-microglobulin mol., and may further comprise an antigen bound to the CD1d binding groove. The invention is further directed to methods of inhibiting or stimulating an immune response with the CD1d-**antibody** compds., in particular anti-**tumor** and autoimmunity responses.

L22 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:213195 CAPLUS
DN 136:114823
TI A comparison of targetting of neuroblastoma with mIBG and anti L1-CAM **antibody** mAb **chCE7**: therapeutic efficacy in a neuroblastoma xenograft model and imaging of neuroblastoma patients
AU Hoefnagel, C. A.; Rutgers, M.; Buitenhuis, C. K. M.; Smets, L. A.; de Kraker, J.; Meli, M.; Carrel, F.; Amstutz, H.; Schubiger, P. A.; Novak-Hofer, I.
CS Department of Nuclear Medicine, The Netherlands Cancer Institute, Amsterdam, 1066 CX, Neth.
SO European Journal of Nuclear Medicine (2001), 28(3), 359-368
CODEN: EJNMD9; ISSN: 0340-6997
PB Springer-Verlag
DT Journal
LA English
AB Iodine-131 labeled anti L1-CAM **antibody** mAb **chCE7** was compared with the effective neuroblastoma-seeking agent 131I-labeled metaiodobenzylguanidine (MIBG) with regard to (a) its therapeutic efficacy in treating nude mice with neuroblastoma xenografts and (b) its **tumor** targetting ability in neuroblastoma patients. The SK-N-SH **tumor** cells used in the mouse expts. show good MIBG uptake and provide a relatively low number of 6,300 binding sites/cell for mAb **chCE7**. **Tumors** were treated with single injections of 131I-MIBG (110 MBq) and with 131I-labeled mAb **chCE7** (17 MBq) and

both agents showed antitumor activity. After therapy with 131I-**chCE7**, the s.c. **tumors** nearly disappeared; treatment with 131I-MIBG was somewhat less effective, resulting in a 70% reduction in **tumor** volume A calculated **tumor** regrowth delay of 9 days occurred with a radioactivity dose of 17 MBq of an irrelevant control **antibody** mAb 35, which does not bind to SK-N-SH cells, compared with a regrowth delay of 34 days with 131I-mAb **chCE7** and of 24 days with 131I-MIBG. General toxicity appeared to be mild, as assessed by a transient, approx. 10% maximum decrease in body weight during the treatments. The superior **growth** inhibition achieved by 131I-**chCE7** compared with 131I-MIBG can be explained by its prolonged retention in the **tumors**, due to slower normal tissue and plasma clearance. Cross-reaction of mAb **chCE7** with L1-CAM present in normal human tissues was investigated by direct binding of radioiodinated mAb to frozen tissue sections. Results showed a strong reaction with normal human brain tissue and weak but detectable binding to normal adult kidney sections. Seven patients with recurrent neuroblastoma were sequentially imaged with 131I-MIBG and 131I-**chCE7**. The results underlined the heterogeneity of neuroblastoma and showed the two imaging modalities to be complementary. 131I-**chCE7** scintigraphy may have clin. utility in detecting metastases which do not accumulate 131I-MIBG, and the **antibody** may hold potential for radioimmunotherapy, either by itself or in combination with 131I-MIBG.

RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d l23 bib abs 1-6

L23 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1
AN 2006:55798 CAPLUS
DN 144:127146
TI Efficient Inhibition of Intra-Peritoneal Tumor Growth and Dissemination of Human Ovarian Carcinoma **Cells** in Nude Mice by Anti-L1-**Cell Adhesion** Molecule Monoclonal **Antibody** Treatment
AU Arlt, Matthias J. E.; Novak-Hofer, Ilse; Gast, Daniela; Gschwend, Verena; Moldenhauer, Gerhard; Gruenberg, Juergen; Honer, Michael; Schubiger, P. August; Altevogt, Peter; Krueger, Achim
CS Klinikum rechts der Isar, Technischen Universitaet Muenchen, Munich, Germany
SO Cancer Research (2006), 66(2), 936-943
CODEN: CNREA8; ISSN: 0008-5472
PB American Association for Cancer Research
DT Journal
LA English
AB The L1 **cell adhesion** mol. is implicated in the control of proliferation, migration, and invasion of several tumor **cell** types in vitro. Recently, L1 overexpression was found to correlate with tumor progression of ovarian carcinoma, one of the most common causes of cancer-related deaths in gynecol. malignant diseases. To evaluate L1 as a potential target for ovarian cancer therapy, the authors investigated the effects of anti-L1 monoclonal **antibodies** (**chCE7** and L1-11A) on proliferation and migration of L1-pos. human SKOV3i.p. ovarian carcinoma **cells** in vitro and the therapeutic efficacy of L1-11A against i.p. SKOV3i.p. tumor growth in nude mice. In vitro, both anti-L1 **antibodies** efficiently inhibited the proliferation of SKOV3i.p. **cells** as well as other L1-expressing tumor **cell** lines (renal carcinoma, neuroblastoma, and colon carcinoma). On two **cell** lines, hyper-crosslinking of L1-11A with a secondary **antibody** was necessary for significant inhibition of proliferation, indicating that crosslinking of L1 is required for the antiproliferative effect. L1-neg. prostate carcinoma **cells** were not influenced by **antibody** treatment. Biweekly treatment of

ovarian carcinoma-bearing mice with L1-11A led to a dose-dependent and significant reduction of tumor burden (up to -63.5%) and ascites formation (up to -75%). This effect was associated with reduced proliferation within the tumors. L1-directed **antibody**-based inhibition of peritoneal growth and dissemination of human ovarian carcinoma **cells** represents important proof-of-principle for the development of a new therapy against one of the leading gynecol. malignant diseases.

RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2
AN 2005:629969 CAPLUS
DN 144:207960
TI In vivo Evaluation of 177Lu- and 67/64Cu-Labeled Recombinant Fragments of **Antibody chCE7** for Radioimmunotherapy and PET Imaging of L1-CAM-Positive Tumors
AU Gruenberg, Juergen; Novak-Hofer, Ilse; Honer, Michael; Zimmermann, Kurt; Knogler, Karin; Blaeuenstein, Peter; Ametamey, Simon; Maecke, Helmut R.; Schubiger, P. August
CS Center for Radiopharmaceutical Science ETH-PSI-USZ, Paul Scherrer Institute, Villigen, Switz.
SO Clinical Cancer Research (2005), 11(14), 5112-5120
CODEN: CCREF4; ISSN: 1078-0432
PB American Association for Cancer Research
DT Journal
LA English
AB Purpose: The L1 ***cell*** **adhesion** protein is overexpressed in tumors, such as neuroblastomas, renal **cell** carcinomas, ovarian carcinomas, and endometrial carcinomas, and represents a target for tumor diagnosis and therapy with anti-L1-CAM **antibody chCE7**. Divalent fragments of this internalizing **antibody** labeled with 67/64Cu and 177Lu were evaluated to establish a **chCE7 antibody** fragment for radioimmunotherapy and positron emission tomog. imaging, which combines high-yield production with improved clearance and biodistribution properties. Exptl. Design: chCE7F(ab')2 fragments were produced in high amts. (0.2 g/L) in HEK-293 **cells**, substituted with the peptide-linked tetraazamacrocycle 3-(p-nitrobenzyl)-1,4,7,10-tetraazacyclododecane-1,4,7,10-tetraacetate-triglycyl-L-p-isothiocyanato-phenylalanine, and labeled with 67Cu and 177Lu. In vivo bioevaluation involved measuring kinetics of tumor and tissue uptake in nude mice with SK-N-BE2c xenografts and NanoPET (Oxford Positron Systems, Oxford, United Kingdom) imaging with 64Cu-3-(p-nitrobenzyl)-1,4,7,10-tetraazacyclododecane-1,4,7,10-tetraacetate-triglycine-chCE7F(ab')2. Results: The 177Lu- and 67Cu-labeled immunoconjugates reached maximal tumor accumulation at 24 h after injection with similar levels of 12%ID/g to 14%ID/g. Blood levels dropped to 1.0%ID/g for the 177Lu fragment and 2.3%ID/g for the 67Cu fragment at 24 h. The most striking difference concerned radioactivity present in the kidneys, being 34.5%ID/g for the 177Lu fragment and 16.0%ID/g for the 67Cu fragment at 24 h. Positron emission tomog. imaging allowed clear visualization of s.c. xenografts and peritoneal metastases and a detailed assessment of whole-body tracer distribution. Conclusions: 67/64Cu- and 177Lu-labeled recombinant chCE7F(ab')2 revealed suitable in vivo characteristics for tumor imaging and therapy but displayed higher kidney uptake than the intact monoclonal **antibody**. The 67Cu- and 177Lu-labeled immunoconjugates showed different in vivo behavior, with 67/64Cu-3-(p-nitrobenzyl)-1,4,7,10-tetraazacyclododecane-1,4,7,10-tetraacetate-triglycine-F(ab')2 appearing as the more favorable conjugate due to superior tumor/kidney ratios.

RE.CNT 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2004:292071 CAPLUS

DN 140:320040
TI 36Fusion proteins comprising CD1d complex, α2 microglobulin and **antibody** or fragment for targeting therapy of **tumor**, autoimmune disease, inflammation and infection
IN Robert, Bruno; Donda, Alena; Cesson, Valerie; Mach, Jean-Pierre; Zauderer, Maurice
PA Vaccinex, Inc., USA
SO PCT Int. Appl., 152 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004029206	A2	20040408	WO 2003-US30238	20030926
	WO 2004029206	A3	20041007		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1413316	A1	20040428	EP 2002-405838	20020927
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
	CA 2502735	AA	20040408	CA 2003-2502735	20030926
	AU 2003275254	A1	20040419	AU 2003-275254	20030926
	EP 1551448	A2	20050713	EP 2003-759526	20030926
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
PRAI	EP 2002-405838	A	20020927		
	WO 2003-US30238	W	20030926		

AB The invention is directed to a compound comprising one or more CD1d complexes in association with an **antibody** specific for a **cell** surface marker. The CD1d complexes comprise a CD1d, a ss2-microglobulin mol., and may further comprise an **antigen** bound to the CD1d binding groove. The invention is further directed to methods of inhibiting or stimulating an immune response with the CD1d-**antibody** compds., in particular anti-**tumor** and autoimmunity responses.

L23 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:407912 CAPLUS
DN 140:213018
TI Targeting of renal carcinoma with 67/64Cu-labeled anti-L1-CAM **antibody chCE7**: selection of copper ligands and PET imaging
AU Zimmermann, Kurt; Grunberg, Jurgen; Honer, Michael; Ametamey, Simon; August Schubiger, P.; Novak-Hofer, Ilse
CS Center for Radiopharmaceutical Science ETH-PSI-USZ, Paul Scherrer Institute, Villigen, CH-5232, Switz.
SO Nuclear Medicine and Biology (2003), 30(4), 417-427
CODEN: NMBIEO; ISSN: 0969-8051
PB Elsevier Science Inc.
DT Journal
LA English
AB In order to optimize radiocopper labeling of anti-L1-CAM **antibody chCE7**, five bifunctional copper chelators were synthesized and characterized (CPTA-N-hydroxysuccinimide, DO3A-L-p-isothiocyanato-phenylalanine, DOTA-PA-L-p-isocyanato-phenylalanine, DOTA-glycyl-L-p-

isocyanato-phenylalanine and DOTA-triglycyl-L-p-isocyanato-phenylalanine). Substitution with more than 11 chelators per **antibody** mol. was found to influence immunoreactivity and biodistributions of 67Cu-MAb **chCE7** significantly. CPTA-labeled **antibody** achieved the best tumor to normal tissue ratios when biodistributions of the different 67Cu-**chCE7** conjugates were assessed in tumor-bearing mice. High resolution PET imaging with 64Cu-CPTA-labeled MAb **chCE7** showed uptake in lymph nodes and heterogeneous distribution in tumor xenografts.
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:213195 CAPLUS
DN 136:114823
TI A comparison of targetting of neuroblastoma with mIBG and anti L1-CAM **antibody** mAb **chCE7**: therapeutic efficacy in a neuroblastoma xenograft model and imaging of neuroblastoma patients
AU Hoefnagel, C. A.; Rutgers, M.; Buitenhuis, C. K. M.; Smets, L. A.; de Kraker, J.; Meli, M.; Carrel, F.; Amstutz, H.; Schubiger, P. A.; Novak-Hofer, I.
CS Department of Nuclear Medicine, The Netherlands Cancer Institute, Amsterdam, 1066 CX, Neth.
SO European Journal of Nuclear Medicine (2001), 28(3), 359-368
CODEN: EJNMD9; ISSN: 0340-6997
PB Springer-Verlag
DT Journal
LA English
AB Iodine-131 labeled anti L1-CAM **antibody** mAb **chCE7** was compared with the effective neuroblastoma-seeking agent 131I-labeled metaiodobenzylguanidine (MIBG) with regard to (a) its therapeutic efficacy in treating nude mice with neuroblastoma xenografts and (b) its tumor targetting ability in neuroblastoma patients. The SK-N-SH tumor **cells** used in the mouse expts. show good MIBG uptake and provide a relatively low number of 6,300 binding sites/**cell** for mAb **chCE7**. Tumors were treated with single injections of 131I-MIBG (110 MBq) and with 131I-labeled mAb **chCE7** (17 MBq) and both agents showed antitumor activity. After therapy with 131I-**chCE7**, the s.c. tumors nearly disappeared; treatment with 131I-MIBG was somewhat less effective, resulting in a 70% reduction in tumor volume A calculated tumor regrowth delay of 9 days occurred with a radioactivity dose of 17 MBq of an irrelevant control **antibody** mAb 35, which does not bind to SK-N-SH **cells**, compared with a regrowth delay of 34 days with 131I-mAb **chCE7** and of 24 days with 131I-MIBG. General toxicity appeared to be mild, as assessed by a transient, approx. 10% maximum decrease in body weight during the treatments. The superior growth inhibition achieved by 131I-**chCE7** compared with 131I-MIBG can be explained by its prolonged retention in the tumors, due to slower normal tissue and plasma clearance. Cross-reaction of mAb **chCE7** with L1-CAM present in normal human tissues was investigated by direct binding of radioiodinated mAb to frozen tissue sections. Results showed a strong reaction with normal human brain tissue and weak but detectable binding to normal adult kidney sections. Seven patients with recurrent neuroblastoma were sequentially imaged with 131I-MIBG and 131I-**chCE7**. The results underlined the heterogeneity of neuroblastoma and showed the two imaging modalities to be complementary. 131I-**chCE7** scintigraphy may have clin. utility in detecting metastases which do not accumulate 131I-MIBG, and the **antibody** may hold potential for radioimmunotherapy, either by itself or in combination with 131I-MIBG.
RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 3
AN 1999:703653 CAPLUS

DN 132:150418
TI Anti-neuroblastoma **antibody chCE7** binds to an isoform of L1-CAM present in renal carcinoma **cells**
AU Meli, Marina L.; Carrel, Francois; Waibel, Robert; Amstutz, Hanspeter; Crompton, Nigel; Jaussi, Rolf; Moch, Holger; Schubiger, P. August; Novak-Hofer, Ilse
CS Center for Radiopharmaceutical Sciences, Paul Scherrer Institute, Villigen, CH-5235, Switz.
SO International Journal of Cancer (1999), 83(3), 401-408
CODEN: IJCNAW; ISSN: 0020-7136
PB Wiley-Liss, Inc.
DT Journal
LA English
AB Immunopptn. after **cell** surface labeling of human neuroblastoma **cells** showed that the anti-neuroblastoma monoclonal **antibody** (mAb) **chCE7** binds to a 200,000 Mr **cell** surface protein. The protein was partially purified by immuno-affinity chromatog. from a human renal carcinoma and a human neuroblastoma **cell** line, which both showed high levels of binding of MAb **chCE7**. NH2-terminal sequences of 18 and 15 amino acid residues were determined Both sequences isolated from the renal carcinoma and the neuroblastoma **cells** showed strong homol. to human **cell adhesion** mol. L1 (L1-CAM), and both were characterized by the NH2-terminal deletion of 5 amino acids, comprising exon 2 of L1-CAM. Reverse transcription-polymerase chain reaction (RT-PCR) anal. of the regions spanning exon 2 and exon 27 of L1-CAM indicated that in neuroblastoma **cells** both transcripts for the full-length and exon-deleted forms are present, whereas in the renal carcinoma **cell** lines only the exon-deleted L1-CAM isoform were detected. Western blot anal. showed that 6 of 7 tested renal carcinoma **cell** lines and 5 of 15 renal carcinoma tissues expressed L1-CAM. In normal adult kidney tissue, very low levels of protein expression were found. Northern blot anal. confirmed that in renal carcinoma and neuroblastoma **cell** lines L1-CAM mRNA levels are correlated with protein expression.
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT